PB85-220572

Exposure and Risk Assessment for Cyanide

Little (Arthur D.), Inc., Cambridge, MA

Prepared for
Environmental Protection Agency, Washington, DC

Dec 81

U.S. Department of Commerce
National Technical Information Service
NTIS

PB85-220572

United States
Environmental Protection
Agency

Office of Water
Regulations and Standards (WH-553)
Washington DC 20460

December 1981
EPA-440/4-85-008

Water

EPA

An Exposure and Risk Assessment for Cyanide

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50272-101

REPORT DOCUMENTATION PAGE	**1. REPORT NO.** EPA-440/4-85-008	**2.**	**3. Recipient's Accession No.** PB85-220572

4. Title and Subtitle
An Exposure and Risk Assessment for Cyanide

5. Report Date Final Revision
December 1981

6.

7. Author(s) Fiksel, J.; Cooper, C.; Eschenroeder, A.; Goyer, M.; Perwak, J.; Scow, K.; Thomas, R.; Tucker, W. and Wood. M.

8. Performing Organization Rept. No.

9. Performing Organization Name and Address

Arthur D. Little, Inc.
20 Acorn Park
Cambridge, MA 02140

10. Project/Task/Work Unit No.

11. Contract(C) or Grant(G) No.
(C) C-68-01-3857
(G) C-68-01-5949

12. Sponsoring Organization Name and Address

Monitoring and Data Support Division
Office of Water Regulations and Standards
U.S. Environmental Protection Agency
Washington, D.C. 20460

13. Type of Report & Period Covered
Final

14.

15. Supplementary Notes

Extensive Bibliographies

16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to cyanide. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in March of 1981.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of cyanide in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to cyanide for various subpopulations.

17. Document Analysis a. Descriptors

Exposure
Risk
Water Pollution
Air Pollution
Effluents
Waste Disposal
Food Contamination
Toxic Diseases
Cyanide

b. Identifiers/Open-Ended Terms

Pollutant Pathways
Risk Assessment

c. COSATI Field/Group 06F 06T

18. Availability Statement Release to Public	**19. Security Class (This Report)** Unclassified	**21. No. of Pages** 129
	20. Security Class (This Page) Unclassified	**22. Pri**

(See ANSI–Z39.18)

See Instructions on Reverse

OPTIONAL FORM 272 (4–77)
(Formerly NTIS–35)
Department of Commerce

EPA-440/4-85-008
March 1981
(Revised December 1981)

AN EXPOSURE AND RISK ASSESSMENT FOR CYANIDE

by

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68-01-5949

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OFFICE OF WATER REGULATIONS AND STANDARDS
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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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Office of Water Regulations and Standards

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ACKNOWLEDGMENTS

The Arthur D. Little, Inc., task manager for this study was Joseph Fiksel. Other major contributors were Charles Cooper (biotic exposure), Muriel Goyer (human effects), Joanne Perwak (human exposure), Richard Thomas and Alan Eschenroeder (environmental fate), Kate Scow (biotic effects), William Tucker (materials balance), and Melba Wood (monitoring data).

1.0 RISK ASSESSMENT SUMMARY

The overall acute risks to humans as a result of the presence of cyanide in the environment appear to be negligible. This is because of the ability of the human to detoxify cyanide rapidly at low exposure levels typically found in the environment. The chronic risks of human exposure are not yet known. On the other hand, there may be significant risks to aquatic biota exposed to cyanide in the vicinity of major point source discharges. The important findings that lead to these conclusions are summarized below.

The major point sources of cyanide releases to water are discharges from Publicly Owned Treatment Works (POTWs), iron and steel production, and the organic chemicals industries. These account for approximately 89% of the estimated 14,000 kkg discharged annually to surface water. The metal finishing and organic chemicals industries account for 90% of the influent to POTWs, so that the metals and organic chemicals industries are the dominant sources of both direct and indirect aqueous discharges. POTW effluents account for about 61 to 71% of direct discharges to water. Another source of direct releases to surface water is non-point runoff from the use of cyanide as an anti-caking agent road salt. The chemical production process for cyanide does not appear to be a significant source of cyanide releases to water.

Emissions of cyanide to air are conservatively estimated to be approximately 20,000 kkg/yr., with over 90% due to automobile exhaust. The resulting background concentrations of hydrogen cycnaide in air would roughly be <65 ng/m^3, assuming that rainout and degradation are relatively slow removal processes. However, based on comparison with carbon monoxide levels from automobile emissions, cyanide concentrations in urban air could frequently be >20 μg/m^3. Rainwater concentrations of cyanide under these conditions would be on the order of 5 μg/l.

Hazards to aquatic organisims occur primarily in the immediate vicinity of a major point source of cyanide. The long-term impact of non-point runoff on cyanide levels in any particular water body is expected to be negligible because the use of road salt is so widely dispersed. However, pulses of non-point runoff due to storm events may result in temporary elevated concentrations. The major fate mechanisms affecting cyanide in water were found to be volatilization and biodegradation. Photolysis may also be an important process in transforming complexed cyanide into free cyanide; however, the rate could not be determined. Therefore, a conservative assumption is that all cyanide discharged was in the free form. Rate constants were estimated for volatilization and biodegradation, and these were applied to cyanide effluents under a variety of assumptions concerning weather, discharge rates, and recieving media. The resulting ambient concentration estimates decreased rapidly as the distance increased from the source. These results implied that cyanide exposure for aquatic life would be highly localized in the vicinity of point source dischargers.

Analysis of STORET surface water monitoring data revealed that only about 40% of observations across the U.S. exceed the EPA criterion of 3.5 µg/l for protection of freshwater life. However, it is probable that waters near cyanide dischargers will show elevated cyanide concentrations. The Pittsburgh area was selected for detailed study of ambient and effluent stations along one river, and increased cyanide levels were noted at locations downstream of several steel plant effluents.

Cyanide is toxic to certain freshwater fish at concentrations of approximately 10 µg/l, with chronic effects being reported at concentrations as low as 5 µg/l. Cold-water fish species appear to be more snesitive than warm-water species, although laboratory results show some exceptions. Aquatic invertebrates were found to be considerably less sensitive than finfish in freshwater. For marine species, existing data are insufficient to estimate absolute toxic levels. In freshwater fish, chronic or sublethal effects generally occur at levels only slightly below the acute LC_{50} levels. This suggests that the chance of adverse effects rises rapidly once the concentration has surpassed a certain species-dependent threshold level.

Because cyanide degrades rapidly in the aquatic environment (half-life on the order of tens of hours), the risks to aquatic life are restricted to within a few river miles of major point sources. However, because of the great variability in experimental conditons and in species sensitivity to environmental stress, the percentage of fish that could die at a certain environmental concentration cannot be accurately predicted. Moreover, because of the sparse nature of ambient monitoring data, it is presently not possible to estimate the percentage of fish that are exposed to potentially toxic levels. Cyanides in sewage do not presently occur at high enough levels to inhibit waste treatment in POTWs. Risks to terrestrial wildlife are expected to be small.

Cyanide's potency as an acute human toxicant is due to its inhibition of respiratory enzymes, resulting in anoxia. However, moderate continuous doses of cyanide can be sustained without ill effects, since detoxification mechanisms are relatively rapid. The human lethal dose of hydrogen cyanide taken orally is believed to be between 50 and 90 milligrams or approximately 1 mg/kg for a 70-kilogram man. The lethal dose of cyanide salts ranges between 200-250 milligrams or approximately 3 mg/kg for a 70-kilogram man. Inhalation of concentrations of hydrogen cyanide $>$ 300 µg/l are fatal within minutes; and inhalation of concentrations of about 90-135 µg/l may be fatal within 30-60 minutes.

Toxicological studies involving the effects of chronic cyanide exposure have been inconclusive. No definitive studies of the carcinogenic, mutagenic or teratogenic/reproductive effects of cyanide have been reported. The only chronic feeding study showed no signs of toxicity during a two-year study period. Although a number of reports have implicated cyanide with several neuropathies, the evidence is not conclusive.

The maximum exposure of the general population from drinking water sources is estimated to be 0.5 mg/day and the exposure of the general population via inhalation is estimated to be about 1.25 ug/day. For those exposed to industrial and automotive emissions, the exposure to cyanide through inhalation may increase to 0.25-1.0 mg/day. All these exposures are insignificant compared with the potential exposure from naturally occurring sources, such as certain foods, and do not appear to represent a significant risk to the general populations.

There are several subpopulations that may be exposed voluntarily to elevated cyanide levels, primarily through the inhalation route. Exposure to cyanide is estimated to range from 0.25 to 18.0 mg/day for subpopulations of 14 million smokers. Exposure for 1,000-20,000 industrial workers could range as high as 70 mg/day, assuming a concentration at the maximum industry standards of 5 mg/m^3. The total exposures for these subpopulations are of the same magnitude as the lethal acute exposure level of 50-90 milligrams. However, the risk of acute effects is not significant because of the long time period of exposure and the rapid detoxification rate. Although these selected subpopulations may experience some risk from chronic exposure, these risks are not quantifiable because of insufficient data on the effects of chronic exposure.

2.0 INTRODUCTION

The Office of Water Regulations and Standards, Monitoring and Data Support Division, the U.S. Environmental Protection Agency, is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated included potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and to estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing a suitable regulatory strategy for reducing the risk, if such action is indicated.

This report provides a brief, but comprehensive, summary of the production, use, distribution, fate, effects, exposure, and potential risks of cyanide. Cyanides are known to be potentially harmful to most living organisms, and are frequently found in the environment in low concentrations due to bbth commercial use and natural occurrence. The purpose of this risk assessment was to quantify the exposure of humans and non-human biota in the U.S. to cyanides, with primary emphasis on water-related exposure routes, and to evaluate the possible health risks associated with such exposure. The technical work described in this report was originally performed in early 1979; the report was revised in mid-1981 to reflect more recent materials balance and monitoring data.

The overall approach followed in this report integrates data on sources, environmental fate, and toxic effects in order to identify significant pathways of exposure and risk (Arthur D. Little, Inc., 1980). Since an assessment must be performed for the nation as a whole, it is necessary to develop observations about the general distribution and impact of a pollutant in the environment. Based upon rates of discharge and of downstream degradation and volatilization, the fate of cyanide discharged into surface water was described. Due to the short half-life of cyanide in water the geographical distribution of this pollutant is far from uniform. Monitoring data, primarily from STORET, were used to determine its environmental distribution and to investigate its presence in the vicinity of several known dischargers by comparison to upstream and downstream concentrations.

The known toxic effects of cyanide are mostly of an acute nature, although possible chronic effects were also investigated for both humans and aquatic life. Therefore, the analysis of potential risks dealt mainly with the likelihood of short-term exposure to concentrations of cyanide in the lethal range. The element cyanide is found in the environment in numerous chemical species. Cyanide occurs most commonly as hydrogen cyanide (HCN), potassium cyanide (KCN), or sodium cyanide (NaCN). Aqueous solutions of cyanide salts will tend to form HCN at a pH of 8 or less, whereas at higher pH cyanide appears mainly in the form of the free ion (CN^-). Cyanide also appears in organic complexes, but this risk assessment has focused upon the presence of free cyanide, either as HCN or CN^-, since this is by far the most toxic form of the substance.

The term "free cyanide" is used to denote cyanide in the form of the simple CN^- anion, which can be destroyed (or measured) by alkaline chlorination. Hence the term "cyanide amenable to chlorination" or "cyanide A." The term "total cyanide" is used herein to describe all forms of cyanide including cyanide complexes that are not readily destroyed by alkaline chlorination. Wherever data permit, the concentration of HCN or CN^- is distinguished from the concentrations of other chemical species.

The report is organized as follows:

Chapter 3.0 presents a materials balance for cyanide that considers quantities of the chemical consumed in various applications, the form and amount of pollutant released to the environment, particularly releases to water, the environmental compartment initially receiving it, and, to the degree possible, the locations and timing of releases.

Chapter 4.0 describes the ultimate distribution of cyanide by considering the physicochemical and biological fate processes that transform or transport cyanide, and by presenting monitoring data for the nation as a whole, as well as for areas in the vicinity of major cyanide dischargers.

Chapter 5.0 considers toxicological effects on and exposure to biota, predominantly aquatic biota.

Chapter 6.0 describes the available data concerning the toxicity of cyanide for humans and laboratory animals and quantifies the likely level of human exposure via major known exposure routes.

Chapter 7.0 presents a range of exposure conditions for humans and other biota and compares these with the available data on effects levels from Chapters 5.0 and 6.0, in order to assess the risk presented by various exposures to cyanide.

REFERENCE

Arthur D. Little, Inc. Integrated exposure risk assessment methodology. Contract 68-01-3857. Washington, DC: Monitoring and Data Support Division, Office of Water Regulations and Standards, U.S. Environmental Protection Agency; 1980.

3.0 MATERIALS BALANCE

3.1 INTRODUCTION

Cyanide discharges to the environment may result during the production of various cyanide compounds or during transportation and use of cyanide compounds, or they may be inadvertently formed, particularly during combustion. In this chapter, the annual releases of cyanide to the environment in the U.S. are estimated. The releases considered include HCN gas to the air, and total cyanides to water and solid waste from major human sources. This risk assessment addresses direct discharges of cyanide to water in greater detail, even though these appear to be much smaller than atmospheric emissions of HCN gas.

The most recent available data were used in deriving emission estimates. Many were based on data specifically for 1976, while others reflect conditions and practices during the mid-1970's. There is no reason to believe that cyanide discharges to the environment fluctuated sharply during that period. Therefore, the estimated environmental releases are believed to be reasonably representative.

3.2 SUMMARY

The production of 177,000 kkg of cyanide compounds and their subsequent (typically inplant) use in the organic chemical industry results in a direct discharge to water of about 1300-1400 kkg/year, 10-14% of the national total of direct discharges and 75% (about 10,000 kkg) of discharges to POTWs. Production of cyanide or cyanide compounds is also estimated to result in 182 kkg/year of acrylonitrile wastes injected into deep wells and approximately 50 kkg/year of solid wastes of complex iron cyanides from the manufacture of iron blue. If properly managed, these wastes should contribute negligibly to cyanide in surface waters.

The production of iron and coke at iron- and steel-making facilities results in the direct discharge of 1407 kkg cyanide per year, or 10-14% of the estimated national total. There are relatively few (less than 100) major coke and blast furnace facilities, and more than 50% of the production capacity is located in 10 major steel-producing urban areas. Therefore, iron and steel production plants are significant local sources. Effluents from blast furnaces also account for about 2% of the cyanide in influent to POTWs.

The magnitude of aquatic discharges from metal finishing operations with cyanide baths is uncertain, but recent data indicate that these discharges comprise about 0.5% (about 65 kkg) of the direct aquatic discharge to the nation's waters and 16% of the cyanide in influents to POTWs. Most (90%) of the cyanides in POTWs are estimated to come from the metal finishing and organic chemicals industry.

The ore mining and dressing industry is estimated to release 2-20 kkg/year or only 0.01 to 0.2% of the national total of cyanide releases, as a result of recent changes in wastewater management practices. However, the relatively large volumes of wastewater handled at only a few mining operations using the froth flotation and cyanidation process may still lead to locally significant discharges.

Cyanide is used as an anti-caking agent in road salt in the form of iron blue, which may be degraded to simple cyanides. Releases from this source to surface water are estimated to total 940 kkg. These are distributed widely yet have been reported to result in high concentrations in surface waters. The collection and disposal of large-volume snow/salt mixtures during street clearing may result in significant contamination of small urban watersheds though there is no evidence that this has resulted in high concentrations of cyanide.

Estimates of discharges from ash ponds at coal-fired power plants are highly uncertain, but available data indicate that ash ponds may be a significant source, both of direct discharge and input to POTWs. Approximately 61-71% of the national load of cyanide to surface waters is discharged from POTWs, making them the largest source type.

Although the chemical form of the cyanide ion, particularly whether it is free or complexed, is significant to its toxicity and chemical behavior, relatively little information is available regarding the ratio of free to total cyanide in major cyanide-containing waste streams. Available analyses for iron and steel effluents indicate that the ratio varies widely (38-90%, Huff & Huff, 1977); 23-36% of the total cyanide at three Chicago POTWs was present as free cyanide.

Table 3-1 presents the most important known man-made sources of cyanide to the environment. The sources of many of the estimates are two reports by Versar, Inc. (1978 a,b), which contain details regarding the rationale behind these estimates. The rationale for items estimated independently, which include production losses, atmospheric emissions, mining effluents, and iron and steel effluents, is discussed below.

The accuracy of most of the numbers in Table 3-1 is ±50% at best, since they are based on national production of capacity figures multiplied by emission factors derived from sources of variable quality and representativeness, rather than on widespread sampling of actual sources. However, the estimates presented for organic chemicals, metal finishing, the iron and steel industry, and POTWs are based on recent sampling and analysis data, and may be used with greater confidence. Estimates of atmospheric emissions by Eimutis _et al_. (1978) are probably conservatively high.

TABLE 3-1. ESTIMATED ANNUAL ENVIRONMENTAL RELEASES OF TOTAL CYANIDE, 1976

Source	Annual Release (kkg/year)				
		Water			
Production and Processes	Air	Direct	POTW	Other	Production or Use (CN^- equivalent)
Hydrogen Cyanide					
via Sohio Process	640^c $0.8\text{-}4.0^b$	0^b	*	182**	87523.1^a
via Andrussow	270^c 0^b	$10\text{-}30^b$	NA		71610.2^a
Sodium Cyanide	*	$<26^b$	NA		14018.0^a
Iron Blue	*	$<10^b$	NA	50-100***	2042.1^a
Zinc Cyanide	*	$<3^b$	NA		932.0^a
Sodium and Potassium Ferrocyanides	*	$<1^b$	NA		853.0^a
Potassium Cyanide	*	$<1^b$	NA		368.0^a
Other Heavy-Metal Cyanides	*	$<1^b$	NA		124.8^a
Uses in Organic Chemicals	1500^c	1322^f	9671^f		159133^a

TABLE 3-1. ESTIMATED ANNUAL ENVIRONMENTAL RELEASES OF TOTAL CYANIDE, 1976 (Continued)

Source	Annual Release (kkg/year)				
		Water			
Production and Processes	Air	Direct	POTW	Other	Production or Use (CN^- equivalent)
Inorganic Chemicals					
Mining Operations	*	2[b], 20[g]	NA		2231.8[a]
Photographic Chemicals	*	2.3[a]	NA		876[a]
Metal Heat Treatment	*	Λ	NA		1585[a]
Pigments	*	*	NA		2995[a]
Metal Finishing	*	65[h]	2041[h]		12112[a]
Anti-Caking Agents	*	940[a]	NA		938[a]
Agricultural Pest Control	62[b]	63[b]	NA		125[a]
Inadvertent Thermally Generated Sources					
Iron and Steel Production	+	1407[i]	294[j]		
Coal Combustion at Steam Power Plants	+	*	*		
Ash Pond Discharge at Steam Power Plants	*	150[a]	950[a]		
Oil Refining	+	12[a]			

TABLE 3-1. ESTIMATED ANNUAL ENVIRONMENTAL RELEASES OF TOTAL CYANIDE, 1976 (Continued)

Source	Annual Release (kkg/year)				Production or Use (CN⁻ equivalent)
		Water			
Production and Processes	Air	Direct	POTW	Other	
Automobile Exhaust	18000[b]	*	*		
Incineration	8-80[b]	*	*		
Cigarette Smoke	6-340[e]				
POTW's	*	6300-9800[b]	NA		
National releases from quantifiable sources	19287-20892	10273-13853			

Notes: *Not expected to be a significant source.
+No data available for quantification, but suspected to be a significant source
ΔNo data available for quantification; cyanide ion is likely to be present in the effluents
**Deep well injection.
***Solid waste to landfill.
NA=not applicable.

[a] Versar, Inc. (1978a)
[b] Arthur D. Little, Inc. estimate
[c] Eimutis *et al*. (1978)
[d] Versar, Inc. (1978b)
[e] Surgeon General (1979)
[f] Versar, Inc. (1981a)
[g] Versar, Inc. (1981b)
[h] Versar, Inc. (1981c)
[i] Versar, Inc. (1981d) (assuming all plants at Best Practicable Technology)
[j] Communication from R. Healy (1981), EPA/MDSD, based on analysis of Versar, Inc. (1981d) and U.S. EPA (1980).

3.3 PRODUCTION

3.3.1 Hydrogen Cyanide

Hydrogen cyanide is produced by two processes (U.S. EPA 1975a, Lowenheim and Moran 1975). In the first process, HCN is produced by reaction of natural gas and ammonia with air (Andrussow Process):

$$2CH_4 + 2NH_3 + 3O_2 = 2HCN + 6H_2O$$

Eimutis et al. (1978) estimated significant air emissions from this process, as shown in Table 3-1. However, in this analysis it was conservatively assumed that all wastes from this process are waterborne (i.e., no air emissions), with raw waste loads of 0.7-2.1 kg/kkg of HCN. The effluent may be treated by alkaline chlorination to give final effluent containing 0.0002-0.005 kg/kkg of oxidizable cyanide (U.S. EPA 1975d) and 0.5 kg/kgg of total cyanide.

In the second process, HCN is a byproduct from the reaction of propylene, air, and ammonia to produce acrylonitrile (Sohio Process):

$$C_3H_6 + NH_3 + 1.5\ O_2 = CH_2 + 2\ HCN + 3H_2O + \text{byproducts (aceto-}$$

nitrile, hydrogen cyanide)

The off-gas from the purification section contains about 2 lb of HCN per ton of acrylonitrile (Monsanto 1973). This is incinerated to destroy HCN before the off-gas is vented to the atmosphere. Assuming a reasonable estimate of 95-99% efficiency, air emissions were estimated at 0.02-0.1 lb of HCN per ton of acrylonitrile (0.01-0.05 kg/kkg). There are no wastewater effluents.

In 1976, about 75% of HCN was produced by the direct process (SRI 1976), leading to the estimated emission shown in Table 3-2.

3.3.2 Sodium and Potassium Cyanide

The alkali metal cyanides are produced by direct neutralization of aqueous HCN with NaOH or KOH, respectively. This is a simple process, which generates no wastes. Water is removed by drying, which provides a potential for some losses during production. Though there are no specific data with which to calculate emissions, the emissions can be estimated by assuming that 10% is lost to effluent and that effluent treatment by alkaline chlorination is 99% efficient (U.S. EPA 1975b). The high efficiency of alkaline chlorination has been demonstrated in metal finishing operations. On this basis the total discharge is estimated to be 0.1% of the 26,420 kkg produced, or 26 kkg, as shown in Table 3-3.

TABLE 3-2. EMISSIONS FROM HYDROGEN CYANIDE PRODUCTION

	kkg/year		
Process/Source	**Direct Process**	**Byproduct Process**	**Total**
Production of HCN	232,000	78,000	310,000
Untreated air emissions	---	78	240-565
Untreated wastewater effluent	162-487	---	
Treated air emissions	---	0.8-4.0	
Treated wastewater effluent			
Total CN	10-30	---	10.8-34
Oxidizable CN	0.1-0.3	---	

Source: Arthur D. Little, Inc.

TABLE 3-3. ESTIMATED ENVIRONMENTAL RELEASES FROM PRODUCTION OF CYANIDE COMPOUNDS, 1976

Compound	Production (kkg)	Aqueous Discharge (kkg)
Sodium Cyanide	26,420	26
Iron Blue	3,740	10[a]
Zinc Cyanide	3,273	3
Sodium and Potassium Ferrocyanides	1,169	1
Potassium Cyanide	935	1
Other Heavy-metal Cyanides	701	1

[a] In the form of complex cyanides.

Source: Arthur D. Little, Inc.

The same assumptions may be used to estimate discharges of other metal cyanides that are produced by similar processes, but in much smaller quantities. Aqueous wastes from the manufacture of sodium and potassium ferrocyanides may contain small amounts of complex cyanides, but quantitative data are not available. About 1-2 kkg are estimated to be released to the environment from these processes.

3.3.3 Iron Blue

The process for iron blue starts with sodium ferrocyanide (i.e., a complex cyanide), ferrous sulfate, and ammonium sulfate. The end product is also a complex and insoluble cyanide. Wastewater effluent is treated to remove most of the cyanide as a solid product; therefore, the small amounts of cyanide that are discharged will be solids that are not removed by the solids separation systems. About 50-100 kkg of complex iron cyanide is contained in the solid waste after treatment, and, assuming good solid separation technology, (i.e., total suspended solids in the waste effluent of 20-30 mg/l) from 1 kkg to 10 kkg of complex cyanide would be discharged to the environment each year.

3.4 AIR EMISSIONS

The major air emissions of hydrogen cyanide and other volatile cyanide-containing compounds occur either from incomplete combustion of fuels in the presence of nitrogen compounds or from chemical processing operations.

3.4.1 Automobile Exhaust

Cyanides have been detected in exhaust gases from automobiles; the average rate of hydrogen cyanide emissions has been reported to be 12 mg/mile (General Motors 1975). The estimated fleet composite emission factor for hydrocarbons in automobile exhaust was 8 g/mile in 1976 (U.S. EPA 1975c). The resultant CN/HC emission ratio (1.5×10^{-3}) multiplied by the total annual hydrocarbon emissions of 12×10^6 kkg/year (U.S. EPA 1978a) yields an estimate of HCN emissions of 18,000 kkg/year.

Applying the above-mentioned ratio to estimates of exhaust emissions compiled by U.S. EPA (1978a), the largest cyanide emissions from automobile exhausts would occur in areas of the highest traffic density, such as California (210 kkg CN/year) or the combined states of New York and New Jersey (1500 kkg tons CN/year). (The fate of such emissions is discussed in Section 4.2.).

Hydrocarbon emissions from vehicle exhaust have been steadily decreasing since 1976. U.S. EPA (1975c) estimated that vehicular emissions of hydrocarbons would decrease to 2.7 gn/mile (66%) reduction by 1985. Assuming that cyanide emissions will be reduced proportionately, significant reductions in cyanide emissions from vehicular exhaust are expected.

TABLE 3-4. ESTIMATED AIR EMISSIONS OF HCN FROM CHEMICAL PROCESSING OPERATIONS

Manufacturing Source		Emissions (kkg/year)
Methyl Methacrylate		1,510
Acrylonitrile (HCN byproduct)		636
Hydrogen Cyanide		274
	Total	2,420

Source: Eimutis *et al*. (1978).

3.4.2 Chemical Processing

Gaseous HCN emissions have been reported for three chemical process industries (Eimutis et al. 1978) and are listed in Table 3-4. The emissions in the table were estimated by considering the various process streams and by carrying out materials balances of the constituents in the streams rather than from actual air emissions measurements. Furthermore, Eimutis et al. (1978) adopted conservative assumptions in the calculations, such as assuming that product losses were all released to the air. In the estimates of waterborne emissions (Section 3.3), some of these losses were attributed to water. Hence, the estimates should be considered as approximations only.

3.4.3 Other Sources

Cyanides are also released into the atmosphere from a number of other sources, such as petroleum refineries, steel mills, and solid waste incinerators. However, very few emission measurements have been carried out and their emission rates have not been estimated.

A potential source of gaseous HCN emissions is the solid waste incineration of cyanide-containing plastics such as acrylonitrile. Approximately 80,000 kkg of cyanide in acrylonitrile are manufactured annually, and eventually all of this becomes solid waste. If one assumes that 5% of all solid waste is incinerated, 4,000 kkg of cyanides in acrylonitrile are being burned annually, not all of which is released into the atmosphere.

Experiments conducted under ideal conditions indicate that the combustion of acrylonitrile releases only 0.2% of the cyanide found in the acrylonitrile. The remainder is apparently converted to carbon dioxide (U.S. Bureau of Mines 1951).

Granted that combustion conditions in solid waste incinerators may be less than ideal, a conservative upper bound estimate for HCN release from acrylonitrile combustion is 2% of the CN contained in the acrylonitrile, leading to an annual release of HCN to the atmosphere from acrylonitrile combustion of 8-80 kkg/yr.

3.5 DISCHARGES TO WATER

3.5.1 Organic Chemical Manufacturing

The estimated release to water in Table 3-1, by Versar, Inc. (1981a), is based on sampling and analysis of effluents from the organic chemical industry. The rate of direct discharge reflects a treatment efficiency of 97.6%, while the indirect discharge total results from an average treatment efficiency of 16.7%. These treatment efficiencies are representative of industry averages based upon a comparison of raw and final effluent loading at the sampled facilities.

The organic chemical industry is difficult to characterize because of the diversity of products and processes, and rapid changes in technology. Most of the facilities in this subcategory are part of complex plants, which are integrated to produce, or use, products that are outside of the conventional industry definition.

There are approximately 2000 direct dischargers in this subcategory and 1750 indirect dischargers. The industry is widely distributed in the eastern half of the United States and on the West Coast, with especially large numbers of facilities in the Delaware River Valley, northeastern New Jersey, and the Houston area.

3.5.2 Metal Finishing

The metal finishing industry, the largest consumer of inorganic CN, uses CN solutions at relatively high concentrations. The metal finishing industry is comprised of numerous small "job" shops and larger volume captive plating shops. Most of the small job shops are assumed to discharge to POTWs.

Los Angeles, Detroit, Providence, and Grand Rapids are the leading cities for metal plating employment with 12, 6, 4 and 3% of the nationwide employment, respectively (U.S. DOC 1976,1977).

Sources of cyanides from metal finishing processes include:

- cleaning solution;
- copper, zinc, brass, silver, and gold plating solutions; and
- metal stripping.

Cyanide concentrations in processing solution range from 0.1 g/l to 1.073 g/l. Process rinse waters and batch dumping of cleaning solution are the major effluent contamination pathways. The average cyanide concentrations in the effluent of 55 shops sampled in the Providence, RI area was 8.4 mg/l, with values ranging from 0.01 mg/l to 44 mg/l (Thibault et al. 1980). Cyanide is present in the effluent as a free ion and/or complexed with metals such as iron, nickel, copper, and zinc. The free ion is readily destroyed by conventional chlorine oxidation treatment processes, while iron and nickel cyanide complexes are stable and require more vigorous oxidizing conditions.

3.5.3 Iron and Steel Making

Three major subcategories of the iron and steel industry discharge cyanide: by-product coke plants, iron making, and sintering. Of these three, cyanide discharges from coking and iron making are much greater than discharges from sintering. The cyanide loadings of Table 3-1 are based on the assumption that all facilities discharge at the BPT (Best Practicable Technology) limitation. If the BAT (Best Available Technology) limitation were achieved, the estimated total direct discharges from the iron and steel industry would be reduced from 1407 kkg/year to 76 kkg/year.

Iron and steel making is concentrated in a few areas of the country. Iron and steel employment is greatest in the following cities: Pittsburgh (16%), Gary (13%), Youngstown (6%), and Chicago (5%) (U.S. DOC 1976, 1977). In a survey of Illinois steelmakers, Huff and Huff (1977) found that three plants discharging to the Calumet River contributed 516 lb/day or about 90 kkg cyanide/year; the fraction of free cyanide present in effluents ranged from 38% to 90%. The Pennsylvania Department of Environmental Resources estimates a total point source loading of 3150 lb/day (520 kkg/yr) in the lower 21 miles of the Monongahela River.

3.5.4 Ore Mining and Processing

In the mineral processing industry, cyanide is used as a solvent in precious metal ore processing and as a reagent in the flotation of copper-moly ores and lead-zinc ores.

3.5.4.1 Cyanidation of Gold-Silver Ores

Cyanidation is standard practice around the world. Cyanide solution is used to dissolve the precious metals, the solution is separated from waste solids, and finally gold or silver is precipitated from the clear solution with zinc dust.

United States companies using this practice in 1976 were: Homestake Mining Co. - Lead, South Dakota; Carlin Gold Mining Co. (Division - Newmont Corp.) - Near Elko, Nevada; Cortex Gold Mines - Near Elko, Nevada (the major operations at this mine closed in 1978); Magma Copper Co., San Manuel Division - Arizona.

3.5.4.2 Flotation of Copper-Moly and Lead-Zinc Ores

In the processing of copper-molybdenite ores, the bulk sulfite flotation concentrate containing iron, copper, and moly-sulfides is processed in a second stage of flotation in which cyanide is used to depress the iron and copper minerals so that the moly can be floated. At least 12 major mines in the U.S. were using this practice in 1976. Most of these mines were in Arizona, with others in Nevada, New Mexico, and Utah.

In processing lead-zinc ores (and copper, lead, zinc ores), cyanide is used to depress the zinc minerals while the lead minerals are floated. Nine major mines in the U.S. were using this process in 1976, located in Colorado, Missouri, Idaho, Utah, and Washington.

3.5.4.3 Releases from Ore Processing

In general, good process control and the retention of mill wastes in tailings ponds to promote oxidation have been adequate for the reduction of cyanide to less than detectable concentrations in the final effluents. Many of the mining operations in which cyanide is used as a solvent or as a reagent have zero discharge; that is, all solution is recycled and reused and nothing is discharged. Zero discharge is defined as Best Practicable Technology (BPT) for the cyanidation process in arid climates (40CFR440.22).

All of the gold-silver cyanidation plants (Homestake, Carline, Cortez, Magma) are reported to have zero discharge. At the Homestake Mine, a $15 million tailings disposal and solution recycle system was completed in 1976 (Sisselman 1976). Operations that use cyanide as a reagent in arid climates have zero discharge. These include essentially all of the mines processing copper-moly ores in Arizona, Utah, Nevada, and New Mexico.

At some of the lead-zinc ore mines and mills, effluent is discharged from tailings ponds. However, when the effluents contain cyanide, they are treated to remove it or reduce its concentration. Cyanide is added as an ore depressant at concentrations ranging from 1-50.4 mg/l (average - 11 mg/l). Below is a list of some lead/zinc mines, their mine capacity, and CN discharges (U.S. EPA 1979):

Mine Location	Ore Processed (kkg/year)	Nature of Discharge	Average [CN] mg/l (# of samples)
Missouri	1,032,000	Treated mine effluent	<0.02 (N = 49)
Missouri	972,300	Secondary pond effluent	<0.02 (N = 2)
Utah	?	Treated effluent	0.06 (N = 5)
Missouri	1,482,000	Final discharge	<0.02 (N = 10)
New York	1,009,100	Lagoon overflow	<0.1 (N = 9)
Idaho	709,000	Treated tailings water (93% treatment efficiency)	<0.03
Maine	209,000	Final treated effluent	<0.02

These data reflect 5,413,000 (37%) of the 14,600,000 kkg of Pb/Zn ore processed at mines that discharge to surface waters in the United States and, therefore, can be regarded as representative of national conditions.

By incorporating the above information with data on water use by mining industries (U.S. Bureau of Census 1972), it is estimated that the annual discharge of CN associated with mining operations is less than 2 kkg/year. Versar, Inc. (1981b) estimated the cyanide discharges from the ore mining and dressing industry at 20 kkg/yr, based on flow-weighted average concentration of 18 μg/l.

3.5.5 Steam-Electric Power Plants

The estimates for ash pond discharge in Table 3-1 are based on analysis of a small number of waste streams, and, therefore, are approximations only. The high combustion temperatures and well controlled combustion conditions in power plant boilers suggest that utility boilers are not a major source of cyanide. Nonetheless, ash ponds have been observed to contain elevated levels of cyanide (U.S. EPA 1978b). There are no analytical data to indicate the degree of complexation of cyanide in ash ponds, but it may be roughly similar to that in iron and steel effluents.

Coal consumption, and corresponding discharge of cyanide from ash ponds, is concentrated in the Appalachian and North Central states. In 1976, 60% of coal produced in the U.S. was burned in Ohio, Pennsylvania, Illinois, Indiana, West Virginia, Kentucky, Michigan, Missouri, North Carolina, and Tennessee (U.S. DOE 1977). Since very sparse data exist on cyanide in power plant waste streams, there is a high degree of uncertainty associated with the estimates in Table 3-1.

3.5.6 Road Salt

Ferrocyanides and iron blue are added to road salt to prevent it from caking. The salt is spread on the road surface during the winter months in the Northern U.S. Virtually all of this salt is probably washed off the roads and into streams and storm sewers. Due to the fact that much of the road salt used in the U.S. is distributed on highways in sparsely populated areas, it is probable that cyanides in road salt are distributed diffusely across the country. The metropolitan areas with the greatest reported usage of road salt are Detroit (27 kkg CN/year); New York (17 kkg CN/yr); Rochester (16 kkg CN/yr); Chicago (13 kkg CN/yr); and Milwaukee (12 kkg CN/yr)(Salt Institute 1975). The CN released from road salt is in the complexed form of ferrocyanide.

3.5.7 POTWs

The discharge of cyanide from POTWs has been estimated by three different methods, all based on data reported by Feiler (1980), compiled from sampling and analysis at 20 POTWs. One estimation approach was to take the average effluent cyanide concentration (210 μg/l) times the total effluent flow rate of all POTWs in the U.S. (34,000 MGD; Marshall 1978) resulting in an estimated cyanide discharge of 9800 MT/year. This approach is based on the assumption that the effluents concentrations at the 20 plants surveyed were representative of all plants across the country. An alternate approach is to use the total cyanide discharged from the 20 plants (169 MT/year) and the fact that the historical flow rates of these plants represent 2.7% of total U.S. POTW effluent flow to estimate a total discharge of 6300 MT/yr. This approach is probably the most accurate if the 20 plants are representative of all plants on a flow capacity basis. Finally, the percent removal of cyanide in the 20 plants was approximately 15%. Then for the total influent to POTWs given by Table 3-1 of 18405 kkg/yr., the resultant discharge of cyanide would be 6400 kkg/yr. The similarity of the discharge estimates is encouraging, but all are based on the assumption that the 20 plants surveyed by Feiler (1980) are representative of all U.S. POTWs. Effluents from three treatment plants in Chicago were analyzed in 1975, and 23-36% of the total cyanide was present as free cyanide.

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4.0 ENVIRONMENTAL DISTRIBUTION

4.1 INTRODUCTION

This chapter provides a link between the estimates of environmental releases of cyanide developed in Chapter 3.0 and the assessment of exposure to cyanide developed subsequently in Chapters 5.0 and 6.0 for aquatic and human receptors, respectively. The discussion considers first the physical and chemical processes that transform and transport cyanide through various environmental media and determine its ultimate distribution. Available data are then presented concerning concentrations of cyanide measured in environmental media.

Because of the short half-life of cyanide in water and air, special consideration is given to the distribution of cyanide in the vicinity of the major sources of releases identified in Chapter 3.0. By use of simple fate models, the behavior of cyanide is profiled for a variety of source and receiving media conditions. Monitoring data are also analyzed for the United States as a whole to determine the distribution of cyanide in surface waters.

4.2 ENVIRONMENTAL FATE

4.2.1 Aquatic Fate

A total of six primary fate mechanisms could contribute to the degradation or reduction of cyanides in water. These are: (1) adsorption onto sediment, (2) complexing with other materials in the water, (3) hydrolysis, (4) photolysis, (5) volatilization, and (6) biodegradation. Four of these mechanisms are addressed below in terms of their significance to cyanide degradation. Since adsorption onto sediment does not occur at a detectable rate (Chester Engineers 1977), this process was eliminated from further consideration. In sunlight, it is likely that photolysis plays a role in breaking down cyanide complexes to form HCN. Because this process has not been documented sufficiently, it is assumed that all cyanide in the water was present as HCN. Thus, an upper limit is placed on the amount of HCN possibly present. As a result of these assessments, only four processes were considered in greater detail: volatilization, hydrolysis, biodegradation, and complexing.

4.2.1.1 Volatilization

The method presented by Southworth (1979) has been used to determine the rate of volatilization of cyanide (HCN) from water. The approach is based on a transfer process across the air-water liquid-film interface.

The rate of volatilization is assumed to be a simple first-order exponential decay with rate constant K_L/depth. K_L is the overall mass-transfer coefficient:

$$K_L = \frac{H\, k_g\, K_1}{H\, k_g + k_1} \quad \text{cmhr}^{-1}$$

Here H is the dimensionless Henry's law constant, assumed to be 0.004 in the calculations, since it is relatively invariant over the temperature range of interest. H represents an equilibrium distribution of the substance between the gas and liquid phases. k_g is the gas phase exchange constant and k_1 the liquid phase exchange constant, both in cmhr^{-1}. The gas-phase exchange constant k_g is a function of wind and current velocity:

$$k_g = [1137.5\ (V_{wind} + V_{current})\ (\frac{18}{\text{Mol. Wt. HCN}})^{\frac{1}{2}}]\ \text{cmhr}^{-1}$$

The liquid phase exchange constant k_1 is also a function of current and wind velocity.

$$k_1 = 23.51\ \frac{(V_{current})}{R^{\,0.673}}\ 0.969\ (\frac{32}{\text{Mol. Wt. HCN}})^{.5} \quad \text{cmhr}^{-1}$$

for $V_{wind} \leq 1.9$ m sec^{-1}, and

$$k_1 = 23.51\ \frac{(V_{current})}{R^{\,0.673}}\ 0.969\ (\frac{32}{\text{Mol. Wt. HCN}})^{.5}\ e^{\,0.526\ (V_{wind} - 1.0)} \quad \text{cmhr}^{-1}$$

for $V_{wind} > 1.9$ m sec^{-1}

where: Mol. Wt. HCN = 1 + 12 + 14 = 27

V_{wind} = wind velocity, m sec^{-1}

$V_{current}$ = current velocity, m sec^{-1}

R = stream depth, m.

The volatilization rate constant K_v can be determined from:

$$K_v = \frac{K_L}{100R}$$

and the equation describing volatilization becomes:

$$n = n_o e^{-K_v t}$$

with: n_o = initial concentraton in water (assuming complete and uniform mixing at all times), mg/l

t = time, hr.

Southworth (1979) points out that "in a given water body, bulk fluid mixing and phenomena such as stratification may play a large role in determining the overall impact of interface controlled processes in removing toxicants. Thus, the approach used yields theoretical maximum volatilization rates, which may be reduced by bulk fluid properties. Other factors which may affect volatilization are the presence of surface films, waves, and aerosol formation."

Dodge and Zabban (1952) conducted experiments in batch quantities to determine volatilization rates of cyanides. Their results generally agree with the method described above when the original experimental data are reduced by the Southworth (1979) method. This supports the validity of the method. In addition, information on H, Henry's constant, shows that the variance of this quantity with temperature is small over the 0°-20°C range.

In the absence of a more precise description of the volatilization process, the rate of volatilization of HCN from water is assumed to equal the rate predicted by the equations above.

4.2.1.2 Hydrolysis

Chester Engineers (1977) provides information on tests conducted to determine the hydrolysis rates of cyanide in river water. River water samples were sterilized to remove biological action. Initial tests were run with sodium cyanide at a concentration of 150 μg/l $(2)^{-1}$ CN, potassium ferri-cyanide at 185 μg/l, and cuprous cyanide at 180 μg/l. Experiments were run at temperatures of 10, 20, and 27°C with a pH of 5, 6, 7, or 8. Some samples were kept in the dark and some were exposed to sunlight. As the pH increased, the rate of degradation of sodium cyanide decreased. At a pH of 5, the rate was 100% greater than at a pH of 8. Cuprous cyanide rates were ~40% less than those for sodium cyanide and potassium ferricyanide. For all three cyanide compounds, the average rate at 10°C was 46% less than at 20°C. The average quasi first-order rate constant at 10°C and at pH between 7 and 8 was 0.0002 hr^{-1} and at 23°C was 0.0033 hr^{-1}.

The following rate model for pH between 7 and 8 was established as a result of the tests for cyanide hydrolysis:

$$\frac{dn}{dt} = -0.0029\ (0.959)^{20-T} n$$

where:

n = concentration, mg/l

T = temperature, °C

t = time, hr.

Integrating the equation for n becomes:

$$n = n_o e^{-0.0029\ (0.959)^{20-T} t}$$

A plot of concentration versus time is shown in Figure 4-1. Half-lives at 0, 10, 20, and 30°C are about 23, 15, 10 and 6.8 days, respectively. At temperatures of interest, times to half concentration as a result of hydrolysis only appear to be in the range of about 10-20 days.

No other data were available to verify this model.

4.2.1.3 Biodegradation

The Chester Engineers (1977) study provides information on experiments investigating the rates of biodegradation of cyanides in river waters. River water samples were taken and microorganisms (bacteria) in this water were initially acclimated to a cyanide and nutrient feed. A selective population of microorganisms capable of degrading cyanide was established. One-gallon samples of river water were obtained and tests were conducted at 4, 7, and 25°C. Sodium cyanide, 50 μg/l, was added to the samples as CN and then inoculated with 1 ml of solution containing acclimated microorganisms. (There are 1.5×10^5 microorganisms/ml or 322 mg/10^{12} microorganisms.) These levels represent actual concentrations in the river. Samples of the test batches were analyzed every 12 hours for cyanide concentration and microorganisms.

Initially, the cells developed slowly until the microoganisms became acclimated to the experimental environment. This involved changing the microorganism population until the organisms capable of deriving energy solely from cyanide became prevalent. Bacteria numbers increased at an exponential rate until the cyanide was completely exhausted; and then they rapidly declined. At this point, 50 mg/l of sodium cyanide as CN were again added to the test batches. Again, growth increased at an exponential rate but with no acclimatization lag. Bacterial counts were determined by the Standard Plate Count method.

As a result of these tests, the following equation was derived to fit the observed data:

$$\frac{dc}{dt} = -0.01\, C^{(1.49-0.0333T)}\, X$$

where: C = cyanide concentration, mg/l

X = microorganism concentration, mg/l

T = temperature, °C

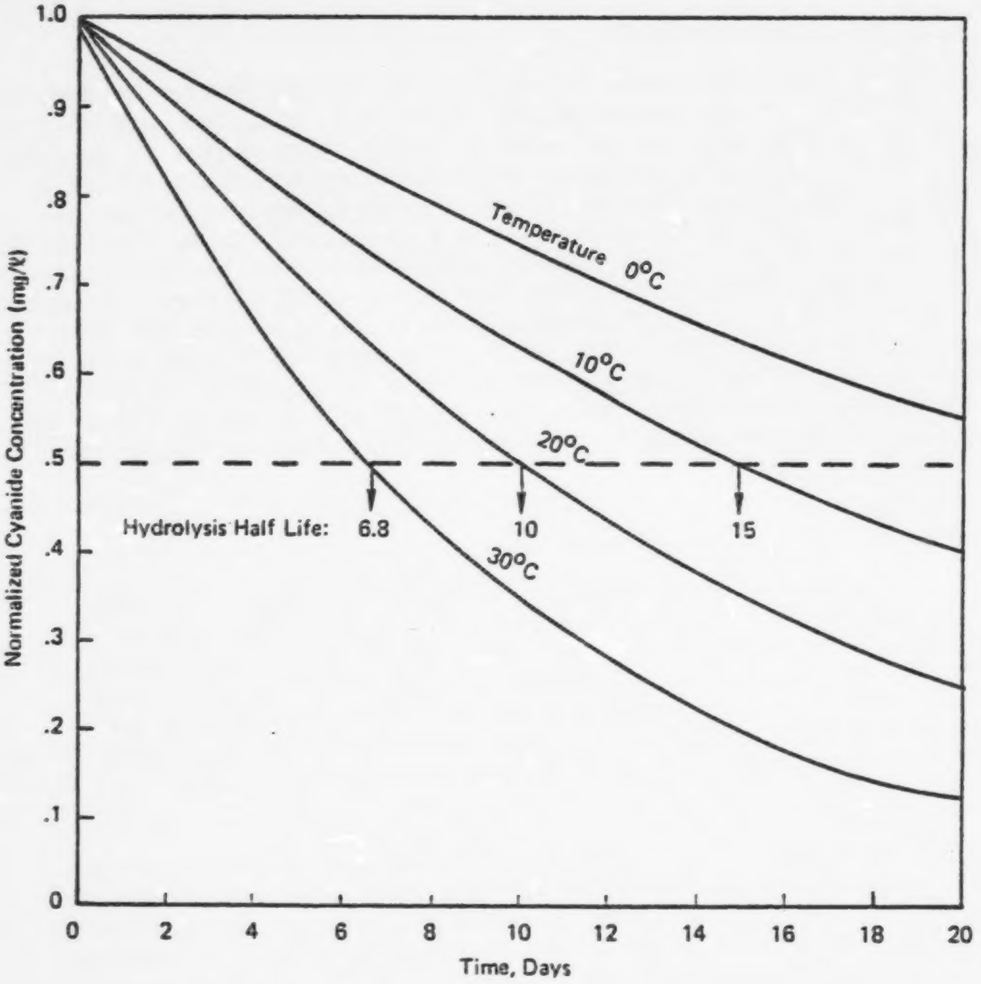
Integrating, the equation for C at any time becomes:

$$C = \left[C_o^{\,1-k} - (0.01Xt)\,(1-k)\right]^{\frac{1}{1-k}}$$

where: t = time, hr.

k = 1.49 - 0.0333T

C_o = initial cyanide concentration, mg/l



Source: Chester Engineers (1977).

FIGURE 4–1 CYANIDE HYDROLYSIS AT TEMPERATURES FROM 0°C TO 30°C FOR pH BETWEEN 7 AND 8 OVER 0 TO 20 DAYS

Depending upon the temperature, half-lives for cyanide range from approximately 10 hours to as much as 60 hours for $1.7 \leq X \leq 7.8$ mg/l of microorganisms.

Growth rates of microorganisms as a result of the cyanide concentration were considered; however, an integrated average concentration of microorganisms in the range shown above was assumed.

4.2.1.4 Cyanide-Iron Complexing

Prober et al.(1977) performed experiments to determine cyanide complexing behavior with iron. Batch runs were made in 4-liter glass vessels with iron salts added to cyanide mixtures in order to bring molar ratios to approximately CN/Fe = 1. Initial cyanide concentrations were between 3.3 mg/l and 11.0 mg/l. The data in Figure 4-2 are for experiments in which the pH ranged between 7 and 12 and temperatures were maintained at about 23°C. Volatilization was shown to be extremely low. Results of the experiments showed that complex formation was very slow for free CN at a concentration less than about 3 mg/l. Cyanide complex formation rates were the most rapid when the pH ranged from 7.5 to 10.5. The data curve from these experiments has been extrapolated several orders of magnitude to show that CN complexing rates at concentrations of CN near those in rivers are extremely slow; these formation rates were anywhere from 10^{-7} mg/l min. to 10^{-15} mg/l min.

4.2.1.5 Fate in the Vicinity of Sources

Based on the preceding sections, both hydrolysis and complexing appear to be considerably slower fate processes than volatilization and biodegradation. Therefore, the latter two processes were considered further in the sample fate calculations. A simple finite difference approach was used to estimate the changes in cyanide concentrations downstream from a point source site. This approach assumed uniform streamflow, complete transverse mixing, and constant discharge rate.

The rate of volatilization depends on such characteristics as the turbulence, depth (ratio of volume-to-surface area), and velocities of the wind and water body being investigated. In addition, the properties of the pollutant in question--specifically molecular weight or diameter and Henry's law constant--affect volatilization rates. It was assumed that depth and flow velocities of the stream and air above it were the major determinants of the rate. The half-life due to volatilization alone is on the order of less than a day to a few days, depending on conditions mentioned above.

Biodegradation is the second mechanism assumed to be operative in determining the fate of cyanides in water. Although many reports in the literature (e.g., Knowles 1976) allude to the degradation of cyanides by microorganisms in natural waters, limited rate data are available for

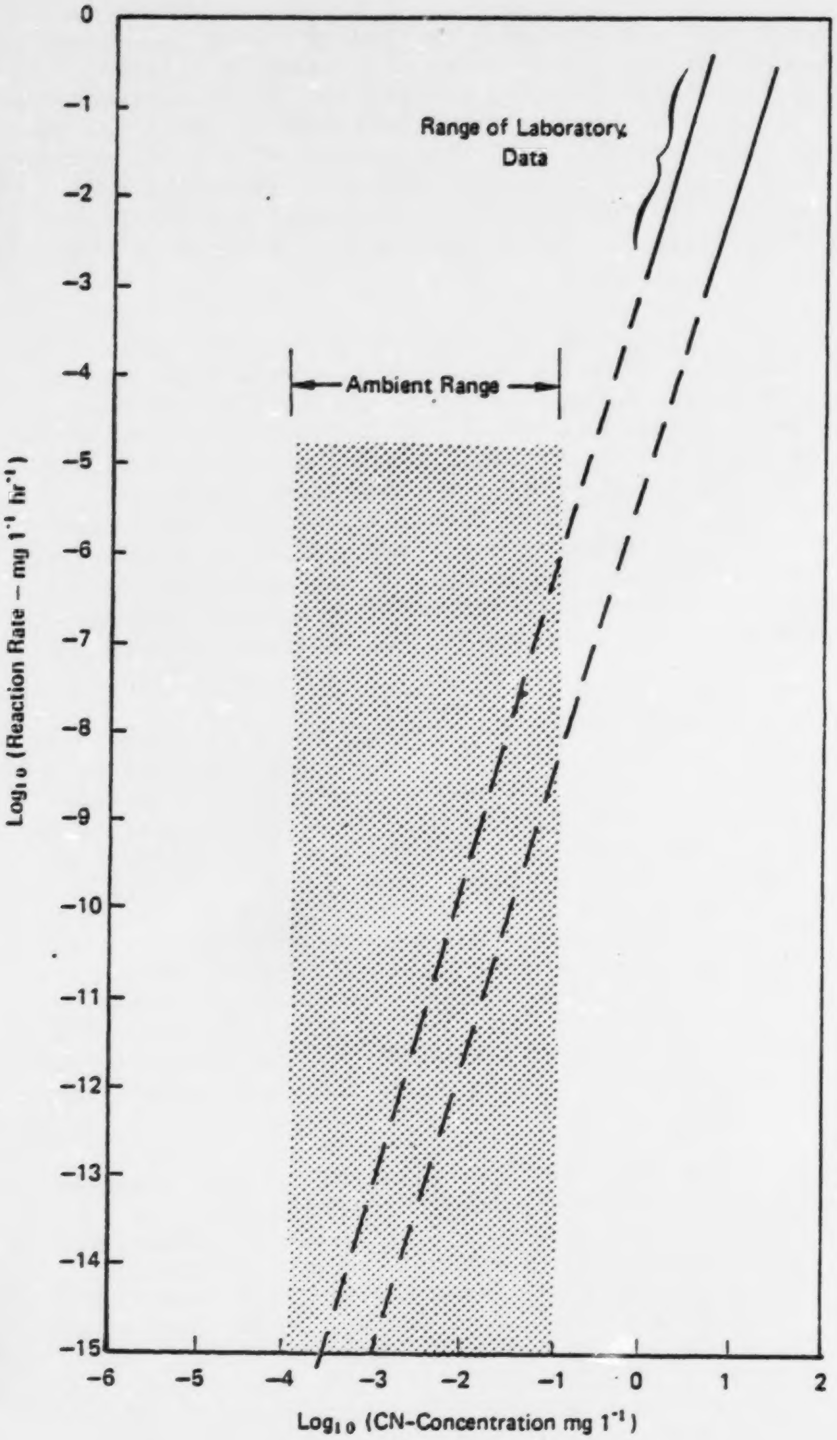


FIGURE 4–2 RATE OF CYANIDE COMPLEX FORMATION WITH IRON AS A FUNCTION OF CYANIDE CONCENTRATION IN WATER (CN/Fe = 1)

Source: Prober *et al*. (1977)

these processes. More data are available concerning biological waste treatment. The results are similar to those of laboratory studies intended to simulate naturally-occurring situations. Here, measurements on river water and acclimated microorganisms were used to construct the water model (Chester Engineers 1977). In the absence of typical concentration data for these organisms in natural waters, a representative value from the laboratory studies was used in the water model calculations. These give half-lives for biodegradation on the order of tens of hours.

The volume of the discharge medium also had to be estimated. Since the fate analysis intends to describe the range of possible situations, several values were assumed. Using data available for flow rates of typical sizes of rivers, a distribution of flow rates for each river was assumed. It was then possible to determine a range of expected conditions. For a maximum concentration case, the receiving medium was assumed to have twice the effluent flow rate, which diluted the concentration in the effluent by a factor of two. The initial concentrations for each of these conditions were determined. Using the stream flow parameters and assumptions about water temperatures and wind speeds, the degradation rates were estimated for cyanide under the modeled conditions.

A further assumption in the fate calculations was that the dilution volume is not changed as it moves downstream; i.e., river dimensions and flow rate remain constant so that no water from other sources is added and no evaporation reduces the initial volume. We assume no concentration gradients in the stream result from differential flow patterns or dispersion plumes downstream of an outfall. Once the effluent was released, it was assumed to be completely and uniformly mixed in the dilution volume. The only modifying mechanisms subsequently operating on the water volume were volatilization and biodegradation. In practice, plume phenomena and topographic irregularities may result in higher cyanide concentrations near a discharge site than those predicted in the model presented here. However, the relative magnitudes of the concentrations estimated under various conditions provide a useful general comparison of situations involving different industrial dischargers.

Detailed results of the fate modeling procedure are not presented here, since many different hypothetical cases were addressed. An example of the results is shown in Figure 4-3. Because the concentrations in individual cases may vary over several orders of magnitude, it is difficult to make a general statement concerning the impact of industry effluents. According to the calculations, cyanide sources did not contribute significantly to high concentrations downstream. The cyanides generally decayed to 50% levels within a few kilometers, although differences in environmental conditions have quite visible effects on the calculated degradation of cyanides. In most cases, the level of cyanide concentration is estimated to drop by an order of magnitude or more within 30 km downstream. At higher initial concentrations and lower wind speeds and water temperatures, the distances

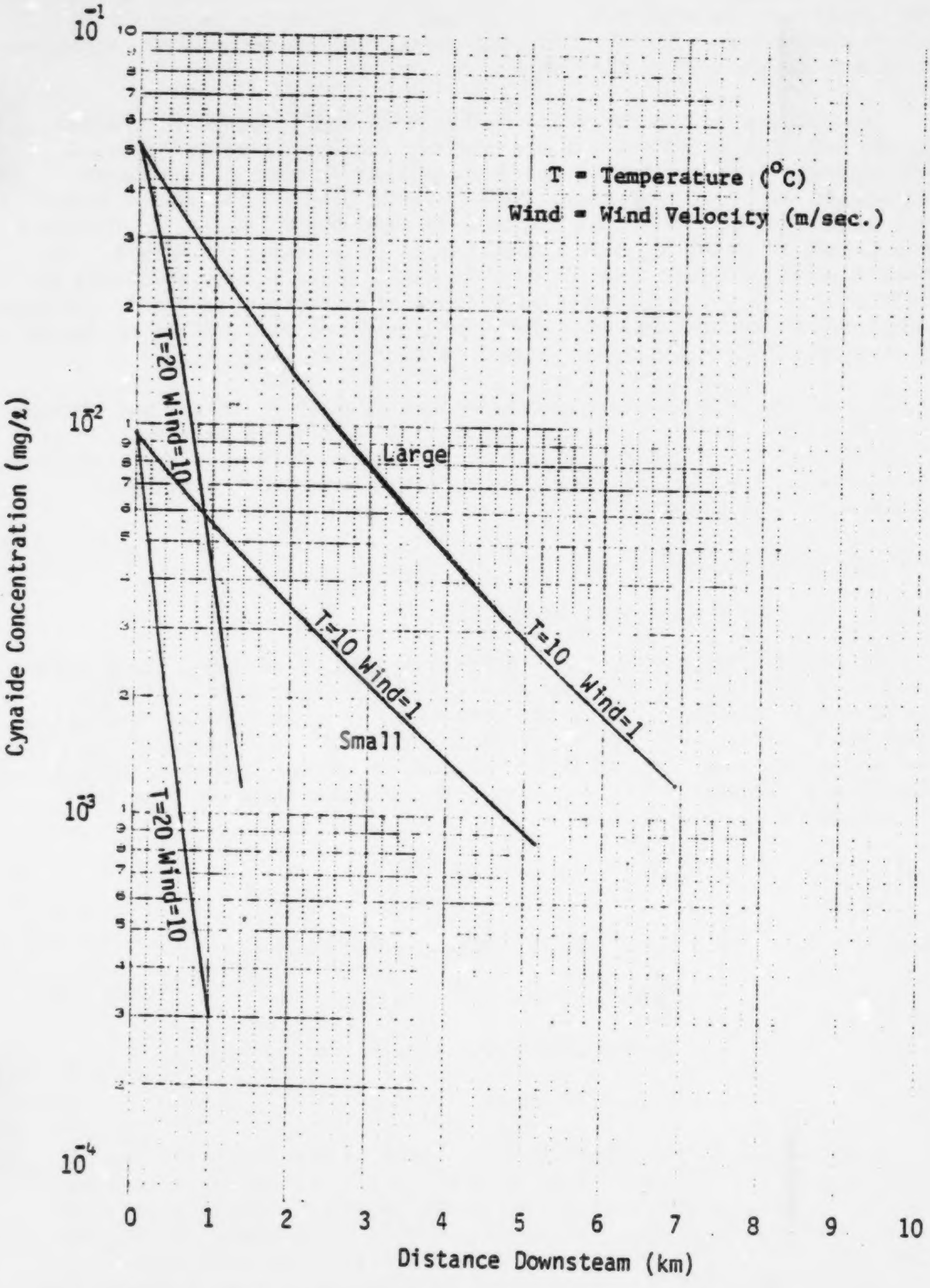


FIGURE 4–3 EXAMPLE OF RESULTS OF FATE MODEL: CYANIDE CONCENTRATIONS DOWNSTREAM OF A SMALL AND LARGE POINT SOURCE ON A SMALL RIVER

may be on the order of 50 km. At lower initial concentrations and higher wind speeds and temperatures, this concentration may be reached 8-10 km or less downstream. For the limiting cases, the characteristic distances are at the higher end of the range.

Temperature is the controlling factor in biodegradation, whereas current and wind speed are the controlling factors in volatilization. Current speed does not have a large variation; in most of the cases modeled, it was about 0.2 m/sec. Wind speeds used in the calculations are 1 m/sec and 10 m/sec. An increase in wind speed lowers the distance tance needed to reach a given concentration by a factor of 4 or 5. An increase in temperature from 10 to 20°C has similar effect, reducing by a factor of 2 to 4 the distance at which a given concentration is reached. An increase in temperature at a low wind speed has more effect on degradation rate than at a high wind speed (a factor of 4 vs. 2).

In summary, elevated concentrations of cyanide in rivers and streams are expected to occur only within 10-30 km of discharge sources. Actual calculation of these concentrations would require site-specific information regarding the initial effluent concentrations and the relevant environmental conditions.

4.2.2 Atmospheric Fate

4.2.2.1 Background Concentrations

The background levels of HCN were estimated on an upper limit basis by assuming that one-half of the entire air emissions (see Section 3.1) is emitted uniformly into the atmosphere over an area approximating a four-sided region with Maine, Virginia, Missouri, and Wisconsin at its corners. A box model (Lucas 1958) was used to obtain concentrations at the downwind boundary of this region. The concentration in mass per unit volume is approximated by:

$$\chi = QS/2HU \text{ for } S >> H$$

where: Q = Mass rate of emissions per unit area

S = Downwind length of the box

H = Mean mixing depth (height of the box)

U = Mean wind speed.

To obtain Q, it is assumed that one-half of the 3.7×10^4 kkg/yr spreads over 1.5×10^6 km^2. S is assumed to be 1500 km and H is 1 km based on the average conditions of a summer morning along the Eastern Seaboard (Holzworth 1972). For these climatic conditions, air trajectories over the entire region average 4.5 m/sec for episode days. (Bach 1975). Assuming no reactions or rainout, the downwind concentration obtained is 0.065 μg/m^3.

The assumption of negligible rainout is reasonable for upper bound calculations because lifetimes of materials with very low vapor pressure exceed a week if absorption on aerosol surfaces is a rate-limiting process (Junge 1977). Residence time for the conditions of the box model is S/U, which is less than 4 days. The concentration in rain drops would be 1.6×10^{-6} μg/l estimated above and the nondimensional Henry's Law constant of 3.95×10^{-3} (mass per unit volume in liquid and gas phases) derived from measured values in the literature at 25°C (Dodge and Zabban 1952).

Reactions of OH-radical with HCN were examined as the principal pathway for potential atmospheric degradation. Because rate constants could not be located in the literature, it was necessary to make estimates based on reaction rates with other hydrogen-bearing compounds and on the relative strengths of hydrogen atom bonding in these compounds. Some of the rate constants (at 300°K) are as follows (Hampson and Garvin 1978):

Reactants	Rate Constant (cm^3/sec)
$OH + C_2H_6$	2.6×10^{-13}
$OH + CH_4$	7.9×10^{-15}
$OH + NH_3$	1.6×10^{-13}

The energies required to remove the first hydrogen atom from each of these compounds are as follows (Schexnayder 1963):

Reaction	Energy (eV)
$C_2H_6 \rightarrow C_2H_5 + H$	4.21
$CH_4 \rightarrow CH_3 + H$	4.40
$NH_3 \rightarrow NH_2 + H$	4.42

The energy for HCN → CN + H ranges from 4.8 eV to 5.6 eV. Further cleavage of the CN Bond to form C + N requires 7.5 to 8.2 eV. Therefore, based on these progressions of bond energies and of reaction rate constants, one would expect an extreme upper limit of, perhaps, 10^{-13} cm^3/sec for the OH + HCN reaction that leads to HCN destruction. An additional reaction producing HOHCN as an intermediate does not involve bond cleavage in the HCN and could proceed at a faster rate than that given by 10^{-13} cm^3/sec. The HOHCN intermediate (if it exists) could readily feed HCN back into the system.

Using the observed range of OH-radical concentrations, the lifetimes of pollutants can be estimated (Eschenroeder et al. 1978). For the rate constant of 10^{-13} cm^3/sec, the lifetime of HCN would vary between the order of a month (in urban atmospheres) to a year in rural atmospheres. Therefore, because both rainout and degradation occur on longer time scales than the residence time in the box model, the conservative assumption for average background concentration of HCN at 0.065 $\mu g/m^3$ is justified.

4.2.2.2 Urban Concentrations

Combustion influences cyanide concentrations in the air of populated areas. Measurements in flame zones indicate that HCN concentrations exceed thermochemical equilibrium levels following the decay of hydrocarbon species in rich hydrocarbon/air combustion (Haynes et al. 1974). The buildup of HCN is postulated to be significant in the chain reactions producing oxides of nitrogen. HCN is believed to be formed by the mechanism:

$$C + N_2 \rightarrow CN + N$$

$$C_2 + N_2 \rightarrow 2CN$$

$$CH + N_2 \rightarrow HCN + N$$

$$M + CN + H \rightarrow HCN + M$$

and CN decays at about 2000°K via:

$$CN + CO_2 \rightarrow OCN + CO$$

$$OCN + H \rightarrow CO + NH$$

$$OCN + H_2 \rightarrow CO + NH_2.$$

In steady-flow combustion, the latter three steps scavenge CN from the system; however, internal combustion engines freeze high temperature equilibrium concentrations into the exhaust gas because of the quenching effect in the rapid cooling as a result of adiabatic expansion during the power stroke.

Consequently, motor vehicles constitute a source of HCN emissions into air that is more significant than steady-flow combustion equipment. Automobiles not equipped with catalytic converters emit 11-14 mg/mi of HCN (U.S. EPA 1978), while catalytically equipped vehicle emissions are on the order of 1 mg/mi under optimal operating conditions. Under malfunction conditions, the catalytically equipped vehicles emit as much (or sometimes several times as much) HCN as the noncatalytically equipped vehicles.

In central cities, the large majority of carbon monoxide in the atmosphere comes from motor vehicles; thus, carbon monoxide is often used as a surrogate for tracing vehicular pollutants. This is especially valuable for tracing HCN, because neither HCN nor CO is significantly affected by smog photochemistry in the scale of urban residence times. One means of circumventing atmospheric modeling is to select a data base of CO ambient measurements and to determine a typical CO/HCN emissions ratio for use as a scaling factor.

The CO monitoring data base was selected from a national tabulation of frequency distributions (U.S. EPA 1977a). Station number 023 in New York City showed some of the highest levels in the nation and was chosen as a prototype worst case. The range of CO/HCN ratios for the Federal Test Schedule emission measurements is 2000-4000 for noncatalyst vehicles. This is based on the 11-14 mg/mi HCN-range (U.S. EPA 1978) and on the 20-60 mg/mi CO range suggested by the standard emission factors tabulations (U.S. EPA 1977b). Vehicles that have malfunctions or maladjustments consistently exhibit ratios of 200-500 (U.S. EPA 1978). Based on these ranges, a representative value of 1000 was assumed for the emissions ratio of CO/HCN.

The application results of the emissions ratio to the New York City Station 023 CO-concentration frequency distribution are shown in Figure 4-4. For comparative purposes, some Bulgarian data on atmospheric concentrations of HCN are indicated on the plot (Kalpasanov and Kurchatova 1976).

The comparability of the measured data for the Sofia, Bulgaria "industrial region" is striking. It is important to note that the New York data are hourly averages and the Bulgarian data are daily averages. Therefore, the factors of difference between the peaks, means, and median reflect not only inaccuracies and emissions variations, but also an expected bias because of the averaging times. For comparative purposes, the "multimedia environmental goal" (MEG) ambient air level of 26 $\mu g/m^3$ (U.S. EPA 1977c) is indicated by a horizontal broken line. (This MEG value was arrived at by an heuristic process and has no averaging time attached to it in the tabulations for ambient media.) Rainwater concentrations in an area of 20-40 $\mu g/m^3$ ambient air concentration would be in the 5-10 $\mu g/l$ range if equilibration occurred during the rainfall.

From these estimates, it may be concluded that significant HCN concentrations in the ambient air occur only in urban areas and that export or rainout are the principal removal mechanisms. Rainwater concentrations are only in the ug/l range, even during high atmospheric loadings of HCN.

4.2.3 Fate in Soil

Because some cyanide may enter the soil such as plants, or from applications and subsequent runoff of road salts, fate in soil was investigated.

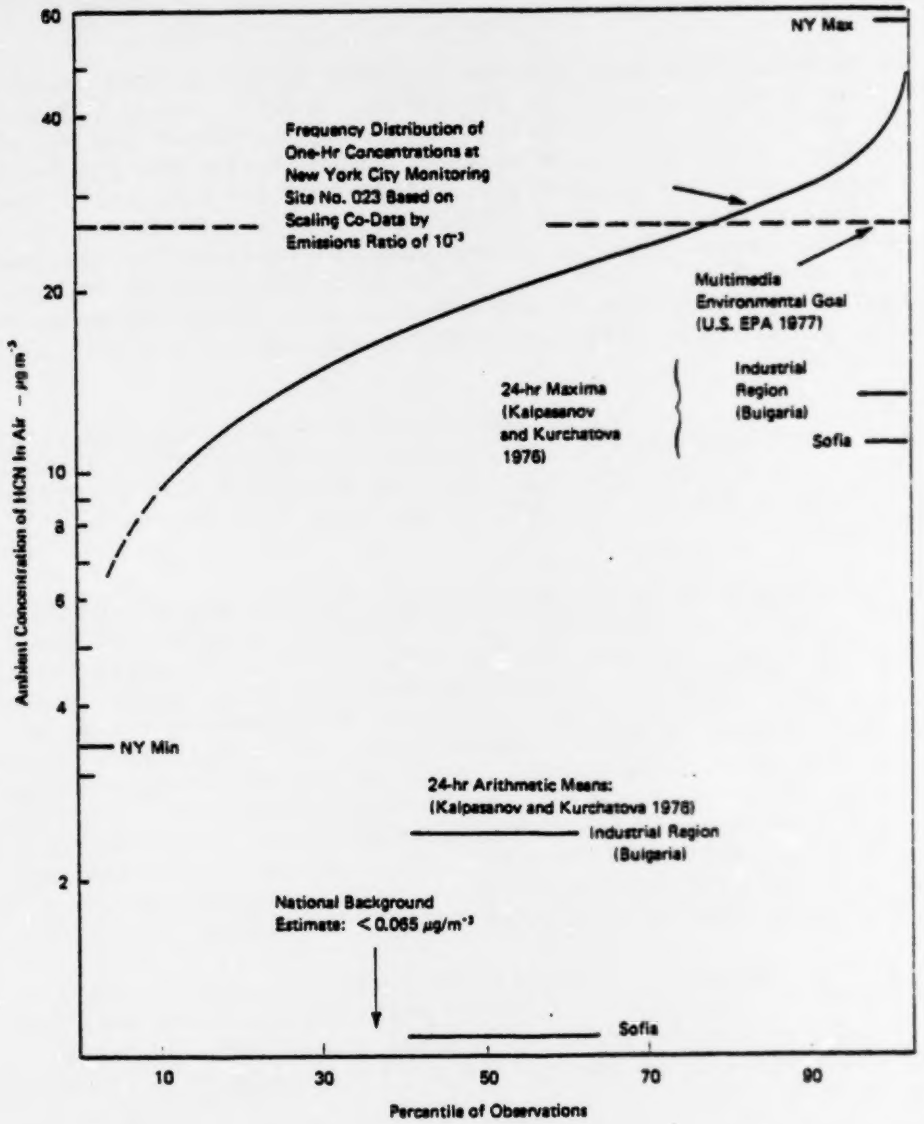


FIGURE 4–4 ESTIMATED AND MEASURED HCN CONCENTRATIONS IN AMBIENT AIR, NEW YORK CITY STATION 023

Rangaswami and Balasubramanian (1963 a,b) found more microorganisms in the roots of cyanide producing plants than in the soil surrounding the plants. When a cyanide solution was added to a suspension containing these microflora, the growth of bacterial microorganisms increased; however, the growth of fungal organisms decreased. Some microorganisms were inhibited 1 or 2 days but were not further inhibited. Fungi added to soils containing cyanide producing plants were not inhibited.

Strobel (1967) tested soil from several sites for their capability to utilize cyanide. All nonsterilized soils were found to have cyanide metabolizing ability, which was attributed to the microorganisms found in the soil. Soil microorganisms were also found to immobilize cyanide nitrogen. Carbon dioxide generation was detected and eventual ammonia (NH_3) fixation utilizing cyanide nitrogen was hypothesized. Strobel (1967) concluded that cyanide carbon and nitrogen were converted to carbonate and ammonia respectively by nonsterilized soils. The soils most able to metabolize cyanide were from areas supporting plants that synthesize cyanide compounds.

Allen and Strobel (1966) contend further that a cyanide cycle exists in nature. Molecules of cyanide could be directly transferred from plants to microorganisms. They could then be transferred by fungi and bacteria back to plants as sources of nutrients without prior conversion to CO_2 and NH_3.

Although these studies indicate that these cyanide cycles occur only in the soils and organisms found in the vicinity of cyanide producing plants, it is reasonable to expect that cyanides will be degraded in soils. Some time may be required to establish the populations capable of degrading the compound. Time required to degrade the cyanides once the population is established is not known, although in Strobel's (1967) study, the ratio of carbon to nitrogen in the soil from degraded cyanide leveled off after about 3 days incubation. Attempts to increase the rate by pretreatment with 10^{-3} molar KCN to acclimate the population were unsuccessful. Cyanide levels lethal to the microorganisms were not stated in any of the references surveyed.

4.3 MONITORING DATA

4.3.1 Introduction

Cyanide proves to be a difficult substance to monitor since it does not persist in the environment. Consequently, discharges into waterways that may be undetected by periodic sampling methods currently used could cause hazards to aquatic life.

The availability of water quality data concerning cyanide has increased in recent years as more monitoring programs are implemented. Of those states with sufficient monitoring data to provide adequate assessments, six states--Illinois, Indiana, Nevada, Ohio, Oklahoma, and Tennessee--reported cyanide water quality problems in 1976 (U.S. EPA 1977d). The exact sources of discharges in these areas were not mentioned in the condensed state reports.

The STORET water quality data system indicates the distribution of ambient concentrations of cyanide; it serves here as the primary source for monitoring data. Two levels of investigation were pursued in retrieving water quality data for cyanide from STORET: national level for the U.S. as a whole, and local level for a selected area. The monitoring results from each are summarized below.

4.3.2 National Monitoring Results

Monitoring of cyanide in ambient waters around the nation aids in pinpointing areas where cyanide concentrations exceed water quality criteria for the protection of freshwater aquatic life and human health.

The U.S. EPA has recommended the following water quality criteria for cyanide (U.S. EPA 1980).

- Freshwater aquatic life - free cyanide criteria to protect freshwater aquatic life: 3.5 μg/l as a 24-hour average and not to exceed 52 μg/l at any time; and
- Human Health - ambient water quality criterion, 200 μg/l (recommended to be identical with existing drinking water standard).

With the mapping capabilities of the STORET Water Quality Control Information System, locations at which the criteria levels are exceeded can be examined. Figure 4-5 displays the 85th percentile of total cyanide concentration as analyzed from 37702 observations recorded at 8750 water quality stations, from 1975 through 1980 (U.S. EPA 1981). Blank areas on the map indicate, for this data set, where monitoring has not occurred. All remarked values were set equal to zero and only stations with a minimum of four observations were included in the analysis.

Data were aggregated by cell sized 30 minutes of latitude by 30 minutes of longitude. Results for total of 682 cells were aggregated in the following concentration ranges:

Concentration Range	No Cells	Percentage
less than or equal to 3.5 μg/l	418	61
greater than 3.5 μg/l and less than or equal to 52 μg/l	238	35
greater than 52 μg/l and less than or equal to 200 μg/l	13	2
greater than 200 μg/l	13	2
	682	100%

Maximum value (for 85th percentile) is 1803 μg/l.

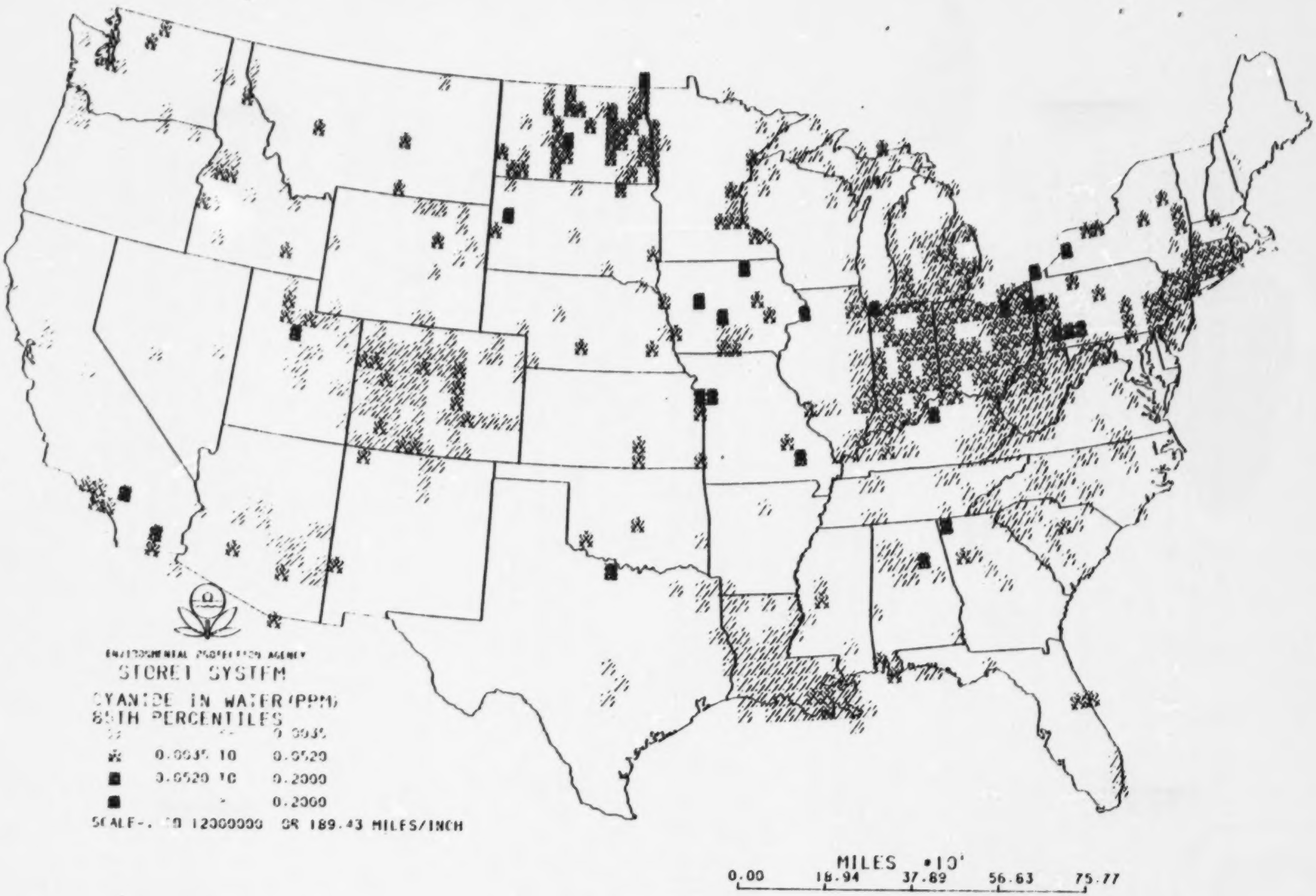

ENVIRONMENTAL PROTECTION AGENCY
STORET SYSTEM
CYANIDE IN WATER (PPM)
85TH PERCENTILES
▨ < 0.0035
✻ 0.0035 TO 0.0520
■ 0.0520 TO 0.2000
■ > 0.2000
SCALE-1 TO 12000000 OR 189.43 MILES/INCH
MILES •10'
0.00
18.94
37.89
56.83
75.77

FIGURE 4–5 TOTAL CYANIDE – 85TH PERCENTILE MAP

Overall, the majority of waters tested nationwide over time do not exceed the lowest criterion of 3.5 μg/l recommended to protect freshwater aquatic life, as a 24-hour average. Thirty-seven of the fifty states (74%), do have some locales with cyanide concentrations in ambient waters greater than 3.5 μg/l. In the latter two ranges, areas in violation of the criteria are fewer in number and identified easily with the map. Areas exceeding the criterion for freshwater aquatic life include portions of southern California, northern Utah, Missouri, Alabama, north central Texas, Kentucky, northwest Indiana, Illinois, Ohio, and western Pennsylvania. Areas with levels exceeding the human health criterion include portions of southern California, North Dakota, South Dakota, Iowa, northwest Georgia, western New York, and western Pennsylvania.

4.3.3 Local Monitoring Results

Because of a heavy concentration of iron and steel operations, the Pittsburgh metropolitan area was selected for an examination of the effect of cyanide discharges on concentrations in local surface water.

A total of 89 ambient stations and 46 effluent stations monitor water quality in the Pittsburgh area. Table 4-1 shows the number of monitoring stations located in each of the counties comprising the metropolitan area. The majority of the stations are located on the Monongahela and Ohio Rivers near major steel operations.

The sampling distribution of mean levels of total CN from all ambient monitoring stations between 1965 and 1979 is exhibited in Table 4-2. Approximately 90% of the stations record mean concentrations below the water quality criterion of 0.2 mg/l for drinking water (NAS 1972). Only 43% of the stations record mean concentrations at or below the recommended limit (0.01 mg/l) for ambient water. This distribution corresponds with the results of the previous analysis of CN levels in geographic areas that contain major CN dischargers.

The sampling distribution of mean levels of total CN from all effluent monitoring stations is shown in Table 4-3. Generally, the effluent stations are located near steel plant outfalls leading to the Monongahela River. Roughly 60% of the stations recorded mean values at or below the mandatory limit for cyanide in the environment.

To observe the impact of cyanide discharges on the aquatic environment in the Pittsburgh area, relationships between ambient and effluent stations were established. Only Allegheny and Beaver Counties reported both ambient and effluent data, which allowed an upstream-downstream comparison. The ratio of ambient to effluent stations is 1.5 to 1 in Allegheny County and nearly 3 to 1 in Beaver County. Figure 4-6 shows the locations of some ambient and effluent monitoring stations along the Monongahela, Ohio, and Beaver Rivers; and the location of medium to large steel plant operations on the Monongahela River.

TABLE 4-1. LOCATIONS OF WATER QUALITY MONITORING STATIONS IN THE PITTSBURGH METROPOLITAN AREA

TYPE OF MONITORING/COUNTY	No Stations
Ambient	
Allegheny County	61
Armstrong County	2
Beaver County	14
Washington County	1
Westmoreland County	11
	89
Effluent	
Allegheny County	41
Beaver County	5
	46

Source: U.S. EPA (1979).

TABLE 4-2. SAMPLING DISTRIBUTION OF AMBIENT MEAN LEVELS OF TOTAL CYANIDE FOR 89 WATER MONITORING STATIONS IN THE PITTSBURGH METROPOLITAN AREA, 1965 to 1979

CN Concentration Range (mg/l)	STATIONS		
	Number	Percent	Cumulative Percent
0.00-0.01	38	43	43
0.02-0.03	27	30	73
0.04-0.05	6	7	80
0.06-0.07	3	3	83
0.08-0.09	1	1	84
0.10-0.22	4	5	89
0.23-0.35	3	3	92
0.36-0.51	0	--	92
0.52-0.67	0	--	92
0.68-0.83	2	2	94
0.84-0.99	1	1	95
1.00+	4	5	100
	89	100.0	

Source: U.S. EPA (1979).

TABLE 4-3. SAMPLING DISTRIBUTION OF MEAN LEVELS OF TOTAL CYANIDE IN INDUSTRIAL EFFLUENT FOR 46 WATER QUALITY STATIONS IN THE PITTSBURGH METROPOLITAN AREA, 1965 to 1979

Concentration Range (mg/1)	STATIONS		
	Number	Percent	Cumulative Percent
0.00-0.01	7	15	15
0.02-0.03	6	13	28
0.04-0.05	5	11	39
0.06-0.07	2	4	43
0.08-0.09	2	4	47
0.10-0.22	5	11	58
0.23-0.35	4	9	67
0.36-0.51	3	7	74
0.52-0.67	3	7	81
0.68-0.83	1	2	83
0.84-0.99	0	--	83
1.00+	8	17	100
	46	100.0	

Source: U.S. EPA (1979).

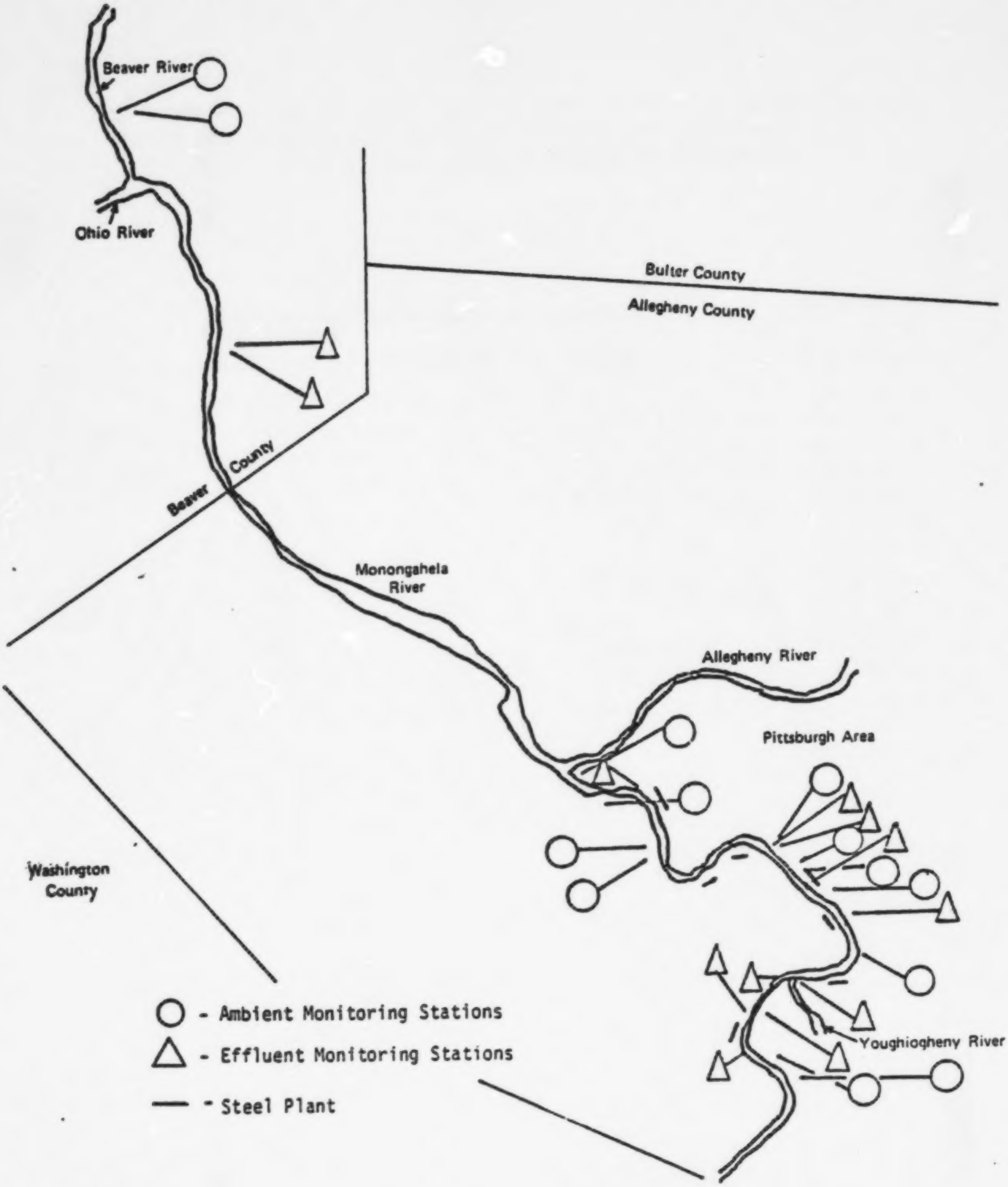

Beaver River
Ohio River
Bulter County
Allegheny County
Beaver County
Monongahela River
Allegheny River
Pittsburgh Area
Washington County
Youghiogheny River
○ - Ambient Monitoring Stations
△ - Effluent Monitoring Stations
— - Steel Plant

FIGURE 4–6 LOCATIONS OF STEEL PLANTS AND WATER QUALITY MONITORING STATIONS ALONG THE BEAVER (OHIO) AND MONONGAHELA (PENNSYLVANIA) RIVERS IN THE VICINITY OF PITTSBURGH

Seven upstream-downstream pairs were established along an 18-mile span of the lower Monongahela River for monitoring stations with common sampling periods. Both ambient and effluent stations tend to be clustered along the river, with outfalls usually about 2 miles apart. Table 4-4 summarizes the data for the seven upstream-downstream pairs. In the case of a cluster of monitoring stations, the range in mean values is shown.

For two of the seven pairs, ambient mean values downstream were higher than the upstream values. The highest effluent values occurred in these two cases and also the downstream ambient stations are within a quarter to one-half mile from the effluent stations at steel plant outfall points. The two relationships represent adjacent steel plant operations. This example suggests that high concentrations may occur within one-half mile of an outfall, but that the cyanide levels downstream will diminish rapidly. The fate analysis in Section 4.2 also supports this conclusion.

TABLE 4-4. UPSTREAM-DOWNSTREAM COMPARISONS OF MONITORED LEVELS OF TOTAL CYANIDE IN SEQUENTIAL ORDER OF LOCATION ALONG THE LOWER MONONGAHELA RIVER

	UPSTREAM			DOWNSTREAM	
Sampling Years	Range in Ambient Mean Values (mg/l)	Miles From Station to Outfall	Range of Effluent Mean Values (mg/l)	Miles From Outfall to Stations	Range in Ambient Mean Values (mg/l)
1971-76	0.02	0.25	0.01 - 0.03	0.50	0.01
1971-75	0.00	0.25	0.13 - 0.14	0.25 - 0.50	0.00
1971-76	0.00 - 0.02	0.50	0.60 - 1.99	0.25	0.02 - 1.05[a]
1971-74	0.02 - 1.05	0.25 - 0.50	0.00 - 1.18	0.50 - 0.75	0.00 - 0.08
1971-77	0.00 - 0.08	1	0.01 - 2.69	0.25 - 0.50	0.02 - 0.74[a]
1971-76	0.02 - 0.74	1	0.24 - 0.81	1.5	0.03 - 0.05
1967-76	0.03 - 0.05	1.5	0.09 - 0.11	0.50 - 0.75	0.00 - 0.02

[a] Cases in which downstream effect was observed.

Source: U.S. EPA (1979).

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5.0 EFFECTS AND EXPOSURE--BIOTA

5.1 EFFECTS ON BIOTA

5.1.1 Introduction

Cyanide is a rapidly acting, highly toxic substance. Most reported research has focused on the acute toxicity of cyanide to freshwater fish. Information regarding long-term effects and bioaccumulation is limited.

This chapter summarizes and discusses the data on the acute effects of cyanide with regard to freshwater and saltwater fish and invertebrate species, sublethal effects data for freshwater fish, and the potential for bioaccumulation. In addition, the relationship between certain environmental conditions and cyanide toxicity is considered. Finally, the minimum concentrations of cyanide in water reported to have adverse effects on various groups of aquatic organisms are presented.

5.1.2 Toxicity to Aquatic Organisms

5.1.2.1 Interpretation of Experimental Results

The toxic effects of cyanide on aquatic organisms that are most commonly described in the literature include the following:

- LC_{50} - concentration lethal to 50% of the population in a stated period of time.
- Reproductive effects - reduction in success of egg hatching, decrease in viability of offspring at various life stages, early or delayed hatching, etc.
- Sublethal effects - alteration of rate of respiration. biochemical changes, organ or tissue damage, (e.g., cell death), inhibition of locomotor activity, etc.
- Bioconcentration factor - concentration in the tissue of an organism divided by the concentration in surrounding water.

Usually cyanides are found in the form of simple alkali cyanides, metal cyanides, or other complex cyanides (U.S. EPA 1977a). The cyanide radical from alkali cyanides, commonly found in industrial wastes, hydrolyzes in an aqueous solution to form free cyanide, which is defined as any combination of HCN (hydrocyanic acid) and CN^- (cyanide ion) (Lind et al., 1977). The predominant fraction between HCN and CN^- depends on the solution pH; when it is <9.0, as is common in natural waters, HCN is considerably more prevalent, and the cyanide ion will be present at lower concentrations.

The toxicity to fish of solutions containing simple cyanides has been primarily attributed to HCN, with CN as a minor contributing factor (Wurhmann and Woker 1948, Bridges 1958, Doudoroff *et al*. 1966). Even the toxicity of metallocyanide complexes has been attributed to HCN (Doudoroff *et al*. 1966), although certain metals may contribute additively or synergistically to the overall toxicity. Since fish commonly live in and thus are tested in water with a pH <9, HCN is more prevalent and consequently highly significant in measured cyanide toxicity. HCN's greater toxicity, however, is also attributed to its relatively lipid-soluble, un-ionized form, which is readily absorbed by aquatic organisms. Charged ions, such as CN^-, are less toxic because of their difficulty in permeating the charged protein surfaces of membranes in exposed areas of the body (Broderius, *et al*. 1977).

During static aquatic toxicity experiments, cyanide levels will decline because of the hydrolysis of CN to HCN and the consequent loss of HCN through volatilization. This loss can have a considerable effect on the results. A significantly greater percentage (100% killed) of minnows died after 14 hours in a freshly prepared cyanide solution than in one that stood for 24 hours (60% killed) before the fish were introduced (Doudoroff 1956). Fish introduced into the solution at 96 hours were not affected at all.

Cyanide will dissipate rapidly from a solution, and thus cyanide toxicity measured by LC_{50} values is usually lower in static water experiments than in flowthrough experiments, in which the cyanide is maintained at a constant concentration. However, reported results from the two kinds of tests differ (only by ≈8% [Herbert and Merkens 1952; Doudoroff *et al*. 1966]), probably because cyanide acts rapidly and may have toxic effects on organisms before it is dissipated. Another contributing factor may be that static conditions lead to low levels of dissolved oxygen in water, which would increase the toxicity of the solution (Doudoroff 1976).

For this risk assessment, the effects of simple cyanides on aquatic organisms are emphasized. Organic cyanides (nitriles) and metallocyanide complexes vary widely in their behavior in water and their toxicity. It is also difficult to determine how significant the cyanide component is in the overall toxic effect observed. The effects data reported here are focused on selected compounds that are dissociated readily to form free cyanide. Unless otherwise noted, the concentrations for simple cyanides reported in Tables 5-1 through 5-3 are concentrations of free cyanide (referring to both the CN^- ion and molecular HCN present).

5.1.2.2 Toxicity of Free Cyanide

Cold freshwater fish were the most sensitive species to cyanide (see Tables 5-1 and 5-2). LC_{50} values ranged from 0.04 mg/l to 0.126 mg/l under flowthrough conditions. The lowest lethal threshold concentration was 0.02 mg/l for the rainbow trout. The majority of the studies indicate that cyanide concentrations > 0.01 mg/l are deleterious to cold water fish.

TABLE 5-1. REPORTED ACUTE EFFECTS OF FREE CYANIDE ON FISH -- FLOWTHROUGH EXPERIMENTS

Concentration (ppm)	Species	Formulation	Effect	Conditions	Source
	COLDWATER FISH				
0.006-.011	Brook Trout (*Salvelinus fontinalis*)	HCN	MATC^a(based on spawning data)	9°-15°C (varied seasonally), pH8, H = 236-239 ppm	Koenst, et. al. (1977)
0.02	"	KCN	27 days - all survived		Karsten (1934)
0.02	Rainbow Trout (*Salmo gairdneri*)		Lethal threshold concentration	3°C	G. B. Ministry of Technology (1968)
0.04-0.05	Brown Trout (*Salmo trutta*)	CN	24 hrs. - LC_{50}	3°C	G. B. Dept. of Environment (1972)
0.05	Brook Trout (*Salvelinus fontinalis*)	KCN	136 hrs. - LC100		Karsten (1934)
0.05	"	KCN	40 days - all survived	~9.5°C	Neil (1957)
0.057	"	CN	Minimum lethal threshold concentration	10°C	Broderius (1977)
0.07	Rainbow Trout (*Salmo gairdneri*)	KCN	74 hrs. - average survival time	17.5°C	Herbert and Merkens (1952)
0.07	Brown Trout (*Salmo trutta*)	KCN	10 days - 40% mortality	15.5°C	Burdick, Dean & Harris (1958)
0.08	Brook Trout (*Salvelinus fontinalis*)	KCN	87 hrs. - LC_{50}		Neil (1957)
0.08	Rainbow Trout (*Salmo gairdneri*)	CN	Lethal threshold concentration	12°-13°C	G. B. Ministry of Technology (1968)
0.1	Rainbow Trout (*Salmo gairdneri*)	NaCN	42 hr - LC_{50}	17°-18°C, pH 7.4-8.0	ORNL/EPA (1978)
0.1	Brown Trout (*Salmo trutta*)	NaCN	Threshold dose - 300 min.	15.6°C	ORNL/EPA (1978)
0.126	Brook Trout (*Salvelinus fontinalis*)	NaCN	288 hrs. - LC_{50}	15.4°C	Cardwell, et. al. (1976)
0.14	Rainbow Trout (*Salmo gairdneri*)	NaCN	Total kill in 27 1/2 hrs.		ORNL/EPA (1978)
0.16	"	NaCN	39.0 min. mean survival time for small fish; 16.0 min. for large fish		ORNL/EPA (1978)
0.5	Brown Trout (*Salmo trutta*)	NaCN	Mean death time = 16 min.	15.6°C	ORNL/EPA (1978)
	WARM WATER FISH				
> 0.056	White Crappie (*Pomoxis annulans*)	KCN	10 hrs.- 100% survival uncertain - (Difficult to compare this study with others)	25°C	Renn (1955)
0.071	Harlequin Fish (*Rasbora heteromorpha*)	CN	Lethal threshold concentration		Abram (1964)
0.086	Smallmouth Bass (*Micropterus dolomieui*)	KCN	Lethal threshold concentration at low oxygen	21°C	Burdick, Dean & Harris (1958)
0.104	"	KCN	Lethal threshold concentration at high oxygen	21°C	"
0.104	Bluegill (*Lepomis macrochirus*)	CN	Minimum lethal threshold concentration	25°C	ORNL/EPA (1978)
0.11	Cichlid (*Cichlasoma bimaculatum*)	NaCN	60 days - no mortality		Brockway (1963)
0.11-0.14	Bluegill (*Lepomis macrochirus*)	KCN	150-300 min. - 50% mortality -(Difficult to compare this study with others)	25°C	Renn (1955)
0.11-0.14	Redbreast Sunfish (*Lepomis auritus*)	KCN	150-350 min. - 50% mortality	25°C	"

TABLE 5-1. REPORTED ACUTE EFFECTS OF FREE CYANIDE ON FISH -- FLOWTHROUGH EXPERIMENTS (Continued)

Concentration (ppm)	Species	Formulation	Effect	Conditions	Source
0.114	Fathead Minnow (*Pimephales promelas*)	NaCN	192 hrs. - LC_{50}	25.3°C	Cardwell, et. al. (1976)
0.116	Bluegill (*Lepomis macrochirus*)	NaCN	168 hrs. - LC_{50}	25.4°C	"
0.120	Fathead Minnow (*Pimephales promelas*)	CN	Minimum lethal threshold concentration	25°C	ORNL/EPA (1978)
0.135	Cichlid (*Cichlasoma bimaculatum*)	NaCN	48 hrs - LC_{50}		Brockway (1963)
0.147	Guppy (*Poecilia reticulata*)	KCN	96 hrs. - LC_{50}	25°C	Anderson (1974)
0.154	Bluegill (*Lepomis macrochirus*)	HCN	48 hrs. - LC_{50}	20°C	Doudoroff, Leduc & Schneider (1966)
0.16	Channel Catfish (*Ictalurus punctatus*)	NaCN	30 hrs. - LC_{50}	25.2°C	Cardwell, et. al. (1976)
0.18	Cichlid (*Cichlasoma bimaculatum*)	Nacn	24 hrs. - LC_{50}	25°C	Brockway (1963)
0.20	Guppy (*Poecilia reticulata*)	CN	50 hrs. - average survival time	18°C	G. B. Dept. of Sci. & Indus. Res. (1956)
0.22	Blacknose Dace (*Rhinachthys atratulus*)	KCN	24 hrs. - LC_{50}	21°C, high oxygen	Lipschuetz & Cooper (1955)
0.236	Guppy (*Poecilia reticulata*)	KCN	theoretical lethal threshold concentration	24°-24.5°C	Chen & Selleck (1969)
0.26	Guppy (*Lebistes reticulatus*)	CN	43 hr. - LC_{50}	24°-25°C pH 7.25	ORNL/EPA (1978)
0.261	Goldfish (*Carassius auratus*)	NaCN	336 hrs. - LC_{50}	25.0°C	Cardwell, et. al. (1976)
0.45	Bluegill (*Lepomis macrochirus*)	KCN	96 hr. - LC50 (medium sized fish)		ORNL/EPA (1978)
0.49	Threespine Stickleback (*Gasterosteus aculeatus*)	NaCN	Killed in 8 hrs.	17°-18°C	"
0.49	Eel (*Anguilla anguilla*)	NaCN	Killed in 12 hrs.	17°-18°C freshwater	"

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[a] MATC = maximum acceptable toxicant concentration.

TABLE 5-2. REPORTED ACUTE EFFECTS OF FREE CYANIDE ON FISH -- STATIC EXPERIMENTS

Concentration (ppm)	Species	Formulation	Effect	Conditions	Source
	COLDWATER FISH				
0.05-0.08	Brook Trout (Salvelinus fontinalis)	KCN	Minimum lethal concentration	8°-10°C	ORNL/EPA (1978)
0.07	Rainbow Trout (Salmo gaironeri)	HCN	48 hrs. - LC_{50}		Brown (1968)
0.09	Brook Trout (Salvelinus fontinalis)	KCN	48 hrs. - LC_{50}	8°-10°C	ORNL/EPA (1978)
0.11	Rainbow Trout (Salmo gairdneri)		Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.2	"		< 3 hrs. - average survival time	18°C	G. B. (1956)
	WARMWATER FISH				
0.04-0.12	Goldfish (Carassius auratus)	KCN	72-96 hrs. - caused mortality		Ellis (1937)
0.06	Cyprinic Species (Leucaspius delineatus)	KCN	2.5 hrs. - lethal threshold concentration	19.5°C	Malacea (1966)
0.074	Harlequin Fish (Rasbora heteromorpha)		168 hrs. - 20% mortality		Abram (1964)
< 0.1	Percid Fish (Acerina cernua)	KCN	100% mortality - LC_{50}	12°C	Gillar (1962)
0.1	Carp (Cyprinus carpio)	CN	24 hrs. - 40% mortality		Silaichuk (1969)
0.1-0.3	Fathead Minnow (Pimephales promelas)	CN	24 hrs or more - LC_{50}		Doudoroff (1956)
0.1-0.3	European Minnow (Phoxinus phoxinus)	CN	24 hrs. or more - LC_{50}		Costa (1965)
0.1-0.3	Mrigal (Cirrhina mrigala)	CN	24 hrs. or more - LC_{50}		Seth et. al., (1967)
0.1-0.3	Green Sunfish (Lepomis cyanellus)	CN	24 hrs. or more - LC_{50}		Lewis and Tarrant (1960)
0.1-0.3	Threespine Stickleback (Gasterosteus aculeatus)	CN	24 hrs. or more - LC_{50}		Costa (1965)
0.13-0.14	Bluegill (Lepomis macrochirus)	KCN	96 hrs. - LC_{50}	30°C	Cairns & Scheier (1963)
0.14	Roach (Rutilus rutilus)	KCN	LC_{50}	12°C	Gillar (1962)
0.15	Bluegill (Lepomis macrochirus)	NaCN	11 hrs. - median survival time	20°C	Broderius (1973)
0.15	"	NaCN	96 hrs. - LC_{50}	25°C	Henderson, Pickering & Lemke (1961)
0.15	"	CN	96 hrs. - LC_{50}		Surber (1965)
0.15		NaCN	96 hrs. - LC_{50}	Hard water	ORNL/EPA (1978)
0.15	Largemouth Bass (Micropterus salmoides)	NaCN	Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.17	Rainbow Darter (Etheostoma caeruleum)	NaCN	Lethal threshold concentration		"
0.17	Pickerel (Esox Americanus vermiculatus)	NaCN	Lethal threshold concentration		"
0.17	Rock Bass (Ambloplites rupestris)	NaCN	Lethal threshold concentration		"
0.17	Bluegill (Lepomis macrochirus)	NaCN	Lethal threshold concentration		"

TABLE 5-2. REPORTED ACUTE EFFECTS OF FREE CYANIDE ON FISH -- STATIC EXPERIMENTS (Continued)

Concentration (ppm)	Species	Formulation	Effect	Conditions	Source
0.17	Bluegill (*Lepomis macrochirus*)	NaCN	≈10 hrs. - LC_{50}		Doudoroff, et. al., (1966)
0.17-0.18	"	KCN	96 hrs. - LC_{50}	18°C	Cairns & Scheier (1963)
0.17-0.23	"	KCN	96 hrs. - LC_{50}	18°-20°C observed rapid decline in some experiments	Cairns & Scheier (1958, 1959, 1963, 1968) Patrick, Cairns & Scheier (1968)
0.2	European Minnow (*Phoxinus phoxinus*)		8 hrs. - average survival time	18°C	G. B. (1956)
0.2	"	KCN	5 hrs. - caused mortality	18.5°-22°C	Malacea (1966)
0.2	Harlequin Fish (*Rasbora heteromorpha*)		4 hrs - average survival time	18°C - Method employed unknown	G. B. (1956)
0.2	Zebra Danio (*Brachydanio rerio*)		12 hrs. - average survival time	"	"
0.2	Guppy		80 hrs. - average survival time	18°C	G. B. (1956)
0.23 0.35	Fathead Minnow (*Pimephales promelas*)	NaCN	96 hr. - LC_{50}'s- more toxic in soft water	25°C - Hard and soft water	ORNL/EPA (1978)
0.23 0.35	"	NaCN	96 hrs. - LC_{50} soft and hard water respectively		Henderson, Pickering & Lemke (1961)
0.24	"	NaCN	48 hrs. - LC_{50}		Black, et. al., (1957)
0.25	Black Bullhead (*Ictalurus melas*)	NaCN	Not lethal in 72 hrs.	24.4°C	ORNL/EPA (1978)
0.25	Golden Shiner (*Notemigonus crysoleucas*)	NaCN	Not lethal in 72 hrs.	24.4°C	ORNL/EPA (1978)
0.26	Yellow Bullhead (*Ictalurus natalis*)	NaCN	Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.26	Pumpkin Seed (*Lepomis gibbosus*)	NaCN	Lethal threshold concentration		"
0.28	Bluegill (*Lepomis macrochirus*)	KCN	24-48 hrs. - LC_{50}	20°C	Turnbull, DeMann & Weston (1954)
0.3	Cyprinid Species (*Leucaspius delineatus*)	KCN	72 hrs - 100% mortality (0% mortality at 0.14 ppm)	12°C	Gillar (1962)
0.31	Goldfish (*Carassius auratus*)	KCN	48-120 hrs. - caused mortality	21.5°C	Powers (1917)
0.33-0.45	Bluegill (*Lepomis macrochirus*)	NaCN	96 hrs. - LC_{50}	18°, 30°C, more toxic at higher temperature. Toxicity not affected by water hardness	ORNL/EPA, (1978)
0.39	Mottled Sculpin (*Cottus bairdi*)	NaCN	Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.40	Minnows (*Unidentified*)	CN	24 hrs. - LC_{50}		Schaut (1939)
0.49	Zebra Danio (*Brachydanio rerio*)	KCN	48 hrs. - LC_{50}	24°C, soft water	ORNL/EPA (1978)
0.5	Goldfish (*Carassius auratus*)	NaCN	72 hrs. - caused no mortality		Bridges (1958)
≤ 0.53	Carp (*Cyprinus carpio*)		Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.53	Goldfish (*Carassius auratus*)	NaCN	48 hrs. - caused mortality	24°-28°C	Bridges (1958)

TABLE 5-2. REPORTED ACUTE EFFECTS OF FREE CYANIDE ON FISH -- STATIC EXPERIMENTS (Continued)

Concentration (ppm)	Species	Formulation	Effect	Conditions	Source
0.53	Mudminnows (*Umbra lima*)	NaCN	Lethal threshold concentration		Michigan Dept. of Conservation (1933)
0.6	Carp (*Cyprinus carpio*)	KCN	72 hrs. - caused mortality		Nehring (1964)
0.64	Mosquito Fish (*Gambusia affinis*)	KCN	24-96 hrs. - LC_{50}	21°-23°C	Wallen, Greer & Lasater (1957)
1.04	Black Bullhead (*Ictalurus melas*)	KCN	28 hrs. - caused mortality		Wells (1916)
1.16	Bitterling (*Rhodeus sericeus amarus*)	KCN	8 hrs. - caused mortality	18.5°-22°C	Malacea (1966)
	MARINE FISH				
0.07	Marine Pin Perch (*Lagodon rhomboides*)	HCN	24 hrs. - LC_{50}	13.7°-20.4°C	Daughtery & Garrett (1951)
0.1-0.3	Eel (*Anguilla japonica*)	CN	24 hrs. or more - LC_{50}		Oshima (1931)
0.1-0.3	Eel (*Anguilla anguilla*)	CN	24 hrs. or more - LC_{50}		Costa (1965)

For fresh, warm-water fish under flowthrough conditions, LC_{50}'s ranged from 0.11 mg/l to 0.45 mg/l (see Table 5-1). The results from static experiments were higher (see Table 5-2); however, all LC_{50}'s were < 1.5 mg/l and the majority were < 0.50 mg/l. Lethal threshold concentrations under flowthrough conditions ranged from 0.071 mg/l to 0.236 mg/l for six species of fish. Under static conditions, the threshold concentrations for eleven species of fish fell generally below 0.5 mg/l, ranging from a very low 0.06 mg/l to 0.53 mg/l. These concentrations should not be considered as true threshold levels and therefore, should not be used to define safe levels of cyanide in water. In many cases, the experiments were not conducted under the controlled conditions necessary to determine the most sensitive concentration. Rather, these studies should be used in conjunction with median lethal concentration (LC_{50}) studies to obtain a general estimate of the toxicity of cyanide to fish.

Little information was available concerning the toxicity of cyanide to saltwater species (see Tables 5-1 and 5-2). Static LC_{50} values for two species of eel were 0.1-0.3 mg/l (Oshima 1931, Costa 1965a). A significantly lower LC_{50} value (0.07 mg/l), also from a static test, was reported for the marine pin perch (Daugherty and Garrett 1951). This compares with the sensitivity of the salmonid species to cyanide under flowthrough conditions. The solution was aerated; thus, high toxicity cannot be attributed to low oxygen supply. The test temperature, however, varied greatly. Consequently, it is difficult to determine whether the pin perch is an extremely sensitive species or whether some condition in this particular experiment (e.g., fluctuating temperature) caused an abnormal response. More study is needed regarding this and other saltwater fish species before the toxicity of cyanide to marine and estuarine biota as a group can be assessed.

Aquatic invertebrates were generally far less sensitive to cyanide than vertebrate species (see Table 5-3). LC_{50} values ranged from 0.4 to more than 3.0 mg/l. All species reported are found in freshwater sytems.

Studies of the sublethal effects of cyanide on fish (see Table 5-4) have generally measured changes in swimming ability, in oxygen consumption, in development of eggs and larvae, and in some biochemical activities. The significance of these changes, especially the nonreproductive type, on fish populations is difficult to determine. A 50% reduction in swimming ability or a decrease in the rate of oxygen consumption may have little impact except during periods of stress, e.g., a period of limited food supply. In a similar way, lower reproductive success may not influence population size except during a year of high mortality when it could have a severe impact. The results of sublethal experiments should be used with acute toxicity results to determine a general (order of magnitude) estimate of the cyanide toxicity to each group of fish.

The reported concentrations of cyanide that caused sublethal effects in fish did not vary radically from lethal levels. Concentrations ranged

TABLE 5-3. REPORTED EFFECTS OF FREE CYANIDE ON AQUATIC INVERTEBRATES

Species	Formulation	Concentration (mg/l)	Effect	Condition	Source
Midge Fly (*Cricotopus bicinctus*)	CN	< 3.2	no effect on survival and maturation	Field study	Surber (1960)
Snail (*Physa heteroclita*)	CN	0.432	96 hrs. LC_{50}	Static	Patrick *et*. *al*. (1968)
Snail (*Physa heterostropha*)	KaCN	1.08	96 hrs. LC_{50}	normal dissolved oxygen	Cairns (1965)
		0.48	"	Low dissolved oxygen	"
Daphnia (*Daphnia magna*)	KaCN	0.4	96 hrs. LC_{50}	Static	Dowden and Bennett (1965)
Lymnacea sp. (egg)	KaCN	130.0	96 hrs. LC_{50}	Static	"
Caddisfly larv. (*Hydropsyche sp.*)	KaCN	2.0	48 hrs. LC_{50}	Static, soft water, 20-22.2°C	Roback (1962)
Mayfly larv. (*Stenonemarubrum*)	KaCN	0.5	48 hrs. LC_{50}	"	"
Daphnia (*Daphnia magna*)	KaCN	0.8	Toxicity threshold 2 days	Static 23°C,	Bringmann and Kuhn (1959)
"	NaCN	< 3.4	Concentration nearly immobilizes	Field study	Anderson (1946)
Amphipod (*Gammarus pulex*)	NaCN	0.49	Survive 3 hrs.	13-14°C Constant flow	Costa (1965b)

TABLE 5-4. REPORTED SUBLETHAL EFFECTS OF FREE CYANIDE ON FISH

Concentration (ppm)	Species	Formulation	Effect	Source
	FRESHWATER FISH			
	Coldwater Species			
0.006-.011	Brook Trout (*Salvelinus fontinalis*)	HCN	MATC (based on spawning data)	Koenst. et. al., (1977)
0.01	Rainbow Trout-juv (*Salmo gairdneri*)	HCN	Induced some degree of hepatic necrosis	Dixon and Leduc (1977)
0.01	Brook Trout (*Salvelinus fontinalis*)	CN	Impaired swimming ability by 75%	Neil (1957)
0.01	Coho Salmon (*Oncorhynchus kisutch*)	NaCN	Impaired swimming ability by 56%	Broderius (1970)
0.01-0.10	Atlantic Salmon (*Salmo salar*)	HCN	Caused damage to developing embryos: effects included delayed hatching, reduced conversion of yolk into body tissue, higher incidence of abnormalities	Leduc (1978)
0.02	Chinook Salmon (*Oncorhynthus tshawytscha*)	CN	Reduction in rate of oxygen consumption	Negilski (1973)
0.02-0.03	Rainbow Trout-juv (*Salmo gairdneri*)	HCN	Reduced mean weight gain by 40%-95%. Also fat gain lower and higher water content	Dixon and Leduc (1977)
0.025	Brown Trout (*Salmo trutta*)	CN	Reduction in rate of oxygen consumption	Carter (1962)
0.05	Brook Trout (*Salvelinus fontinalis*)	CN	Impaired swimming ability by 65%	Neil (1957)
	Warm Water Species			
0.019	Fathead Minnow (*Pimephales promelas*)	HCN	Egg production significantly reduced	Lind, et. al., (1977)
0.044	"		Egg hatchability significantly reduced	"
0.06	Cichlid (*Cichlasoma bimaculatum*)	CN	Impaired swimming ability	Brockway (1963)
0.09	"	NaCN	Impaired swimming ability	Leduc (1966)
0.09-0.1	"		Fin damage	Leduc (1966)
0.10	"	NaCN	Affected enzyme activity in liver	Brockway (1963)
0.10	"			"
1.6	Mummichog (*Fundulus heteroclitus*)		Slowed development of embryos in late embryonic stages	Philips (1940)
	MARINE FISH			
0.33	Cunner (*Tautogolabrus adspersus*)		Development of later stages delayed	Philips (1940)
0.65	"		Development delayed and disintegration of embryos after several hours	"
0.78	Threespine Stickleback (*Gasterosteus aculeatus*)		Reduction in rate of oxygen consumption	Jones (1947)
2.6	Cunner (*Tautogolabrus adspersus*)		Development ceased	Philips (1940)

from 0.01 mg/l to 0.10 mg/l for cold-water species and 0.019-1.6 mg/l for warm-water species. Again, certain effects, such as reproduction, varied considerably among species. Swimming ability was affected in several species, however, at approximately the same concentration.

5.1.2.3 Toxicity of Other Cyanide Compounds

The reported effects of selected non-metal cyanide complexes on aquatic organisms are presented in Table 5-5. The most toxic of the organic compounds (and other miscellaneous forms) were cyanogen chloride, lactonitrile, and malononitrile. For these compounds, LC_{50}'s were <1 mg/l. Their toxicity was similar to that of a simple cyanide solution with the same concentration of free cyanide. For many of the other organic compounds, effects were observed at much higher concentrations. This may result from their greater recalcitrance to hydrolysis and any other reactions that liberate and make available the free cyanide portion of the compound to organisms. The other cyanide ions, thiocyanate and cyanate, were also reported as being much less toxic than free cyanide (Doudoroff 1976).

It is difficult to generalize about the toxicity of metal cyanide complexes. The effect of the free cyanide fraction and/or the metal ion can determine toxicity. In turn, these effects are influenced by solution pH and other environmental conditions, synergistic interactions and the compound's stability (e.g., its solubility, photodegradability, etc.).

Concentrations of selected metal cyanide compounds that have been reported to have toxic effects on aquatic organisms are presented in Table 5-6. These results have been selected only to provide an indication of the concentration ranges of cyanide compounds with lower toxic levels that affect aquatic organisms.

The toxicity of some of the less toxic cyanide complexes of metal ions (e.g., nickel and iron) appear to depend on molecular HCN content (Doudoroff 1966). Other metal cyanide complexes--silver, zinc, and copper--were more toxic than other complexes, apparently because of the higher toxicity of the ion present. In all test cases, except one, effective concentrations were higher than those of simple cyanides tested under the same conditions (Lipshuetz and Cooper 1955, Doudoroff 1956). The exception was an experiment reporting zinc-cyanide and cadmium-cyanide complexes as more toxic than simple cyanide, suggesting slight synergistic activity (Doudoroff 1956). Another study on zinc contradicted these results (Cairns and Scheier 1968). In this case, the zinc-cyanide complex was less toxic. Not enough information about this assessment is available to make any conclusions about the relative toxicities of zinc- and cadmium-cyanide complexes and free cyanide.

In general, free cyanide appears to be the most toxic form of cyanide in water. Any conclusions or decisions based on effects concentrations of free cyanide, therefore, would also cover the effects of most cyanide compounds.

TABLE 5-5. REPORTED EFFECTS OF ORGANIC AND OTHER CYANIDE COMPOUNDS ON FISH

Concentration (ppm)	Species	Compound	Effect	Conditions	Source
< 0.1 (0.04 CN)	Rainbow Trout (*Salmo gairdneri*)	Cyanogen chloride	Lethal threshold concentration	Static, 17°-20°C	Allen, et. al., (1948)
0.22 (0.08 CN)	Marine Pin Perch (*Lagodon rhomboides*)	Lactonitrile	24 hr. LC_{50}	Static, 13.7°-20.4°C	Daughtery & Garrett (1951)
0.5	Rainbow Trout (*Salmo gairdneri*)	Malononitrile	72 hr. LC_{50}		G. B. (1973)
0.51 (0.19 CN)	Bluegill (*Lepomis macrochirus*) White Crappie (*Pomoxis annularis*) Golden Shiner (*Notemigonus crysoleucas*)	Lactonitrile	Fatal in 10 hrs.	Flow through, 25°C	Renn (1955)
0.71 (~0.25 CN)	Fathead Minnow (*Pimephales promelas*)	Lactonitrile	96 hr. LC_{50}	Flow through, soft water, 25°C	Henderson, et. al., (1961)
2.6 (1.3 CN)	"	Acrylonitrile	30 day LC_{50}	Flow through, soft water, 25°C	"
10.1	"	Acrylonitrile	96 hr. LC_{50}	Flow through, soft water, 25°C	"
11.8	Bluegill (*Lepomis macrochirus*)	Acrylonitrile	96 hr. LC_{50}	Static, soft water, 25°C	"
25 (12 CN)	Marine Pin Perch (*Lagodon rhomboides*)	Acrylonitrile	24 hr. LC_{50}	Static, variable temperatures	Daughtery & Garrett (1951)
33.5	Guppy (*Lebistes reticulatus*)	Acrylonitrile	96 hr. LC_{50}	Static, soft water, 25°C	Henderson, et. al., (1961)
56	Mosquito Fish (*Gambusia affinis*)	Ammonium Thiocyanate	All fish died in 144 hrs.	Static, 16°-23°C	Wallen, et. al., (1957)
75 (30 CN)	Creek Chub (*Semotilus atromaculatus*)	Sodium Cyanate	LC_{50}		Washburn (1948)
78	Bluegill (*Lepomis macrochirus*)	Benzonitrile	96 hr. LC_{50}	Static, soft water, 25°C	Henderson, et. al., (1961)
78, 135	Fathead Minnow (*Pimephales promelas*)	Benzonitrile	96 hr. LC_{50}	Static, 25°C, hard & soft water respectively	Henderson, et. al., (1961)
114	Mosquito Fish (*Gambusia affinis*)	Ammonium Thiocyanate	96 hr. LC_{50}	Static, 16°-23°C	Wallen, et. al., (1957)
400	Guppy (*Lebistes reticulatus*)	Benzonitrile	96 hr. LC_{50}	Static, soft water, 25°C	Henderson, et. al., (1961)
720	Bluegill (*Lepomis macrochirus*)	Adiponitrile	96 hr. LC_{50}	Static, soft water, 25°C	"
775	Guppy (*Lebistes reticulatus*)	Adiponitrile	96 hr. LC_{50}	Static, soft water, 25°C	"
820, 1250	Fathead Minnow (*Pimephales promelas*)	Adiponitrile	96 hr. LC_{50}	Static, 25°C, soft and hard water respectively	"
1000-1850	Fathead Minnow, Guppy and Bluegill	Acetonitrile	96 hr. LC_{50}'s	Static, 25°C, soft water	"
3600-4450	Fathead Minnow, Guppy and Bluegill	Oxydipropionitrile	96 hr. LC_{50}'s	Static, 25°C, soft water	"

TABLE 5-6. REPORTED EFFECTS OF METAL CYANIDE COMPOUNDS ON FISH

Compound	Concentration (ppm) as CN	Species	Effect	Source
Zinc - and Cadmium - Cyanide Complex	0.23 (NaCN)	Fathead Minnow (Pimephales promelas)	96 hr. LC50's. Indicated slight synergistic activity	Doudoroff (1956)
	0.18 (NaCN + $ZnSO_4$)			
	0.17 (NaCN + $CdSO_4$)			
	0.40-0.64	Bluegill (Lepomis macrochirus)	LC$_{50}$'s of metal cyanide complexes slightly greater than those predictable on basis of determined molecular HCN levels	Doudoroff, et. al., (1966)
	0.18 (KCN)	Bluegill (Lepomis macrochirus)	96 hr. LC$_{50's}$	Cairns & Scheier (1968)
	0.26 (KCN + $ZnCl_2$)			
Nickel-Cyanide Complex	0.42 (pH 6.5-6.6)	Fathead Minnow (Pimephales promelas)	96 hr. LC$_{50}$. Concluded that acute toxicity dependent on molecular HCN content	Doudoroff (1956)
	730.0 (pH 8.0)			
	0.95 (NaCN + N SO_4)	Bluegill (Lepomis macrochirus)	96 hr. LC$_{50}$	Doudoroff (1956)
Silver-Cyanide Complex	10 at pH 7.5 (0.02 ppm HCN), pH 6.5 (0.12 ppm HCN) and pH 6.0 (0.19 ppm HCN)	Bluegill (Lepomis macrochirus)	Median resistance time at pH 6.5 and 7.5 very similar. Much more rapid toxic effects at pH 6.0. Concluded that silver ion itself has high toxicity	Doudoroff, et. al., (1966)
	6.0 (freshwater) 3.0 (seawater)	Threespine Stickleback (Gasterosteus aculeatus)	24 hr. LC$_{50}$	Broderius (1973)
	10.0	Bluegill (Lepomis macrochirus)	24 hr. LC$_{50}$	Broderius (1973)
	< 7.0	"	96 hr. LC$_{50}$	"
Copper-Cyanide Complex	0.22 (KCN)	Western Blacknose Dace (Rhinichthys atratulus)	24 hr. LC$_{50}$'s. Toxicity decreased with increased copper. Test solutions were not aged to point of equilibrium	Lipshuetz and Cooper (1955)
	0.38 (KCN + CuCN w/mole ratio CN/Cu of 4.0)			
	0.47 (ratio 3.75)			
	0.71 (ratio 3.0)			
	0.25 (NaCN)	Fathead Minnow (Pimephales promelas)	24 hr. LC$_{50}$'s. Indicated that complexing decreased availability of both compounds	Doudoroff (1956)
	0.25 ($CuSO_4$)			
	2.2 (NaCN + $CuSO_4$)			
Iron-Cyanide Complex	0.26 (KCN)	Eel (Anguilla japonica)	Both iron-cyanide complexes were lethal in same period of time as KCN.	Oshima (1931)
	3.9 ($K_4Fe[CN]_6$ or $K_3Fe[CN]_6$)			
	0.85 ($K_4Fe[CN]_6$)	Blacknose Dace (Rhinichthys atratulus) Creek Chub (Semotilus atromaculatus) Silvery Minnow (Hybognathus regius)	Fatal in 1-1^1/2 hrs when solutions exposed to light. When solutions kept in dark, lethal concentration considerably higher (~1700 ppm)	Burdick and Lipschuetz (1950)
	300 ($K_4Fe[CN]_6$ kept in dark)	Rainbow Trout (Salmo gairdneri)	24 hr LC$_{50's}$	Bucksteeg (1961)
	30 (" partially illuminated)			
	2 (" exposed to full light)			
	500 ($K_3Fe[CN]_6$ kept in dark)	Bluegill (Lepomis macrochirus)	Fish survived > 48 hrs. in $K_3Fe[CN]_6$ solution but median survival time was 145 minutes in $K_4Fe[CN]_6$ solution. Attributed higher toxicity of latter compound to greater instability (dissociation to HCN)	Broderius (1973)
	500 ($K_4Fe[CN]_6$ kept in dark)			

5.1.2.4 Bioaccumulation

Cyanide does not appear to bioaccumulate in aquatic organisms (ORNL/U.S. EPA 1978). If the cyanide concentration in water is not high enough to kill an organism, cyanide is metabolized and discharged. In aquatic systems, biodegradation is thought to be a predominant fate, of equal importance to hydrolysis (Doudoroff 1976). If this is the case, residues of cyanide found in fish tissue would indicate that the substance had only recently entered the aquatic system.

5.1.2.5 Influence of Environmental Factors

The relationship between cyanide toxicity and water temperature is not clear. At relatively high concentrations (0.3-1.0 mg/l as CN), cyanide is lethal more rapidly at higher temperatures, with an inversely proportional relationship between temperature and the logarithm of time to death (Wuhrmann and Woker, 1953, 1955, Sumner and Doudoroff 1938). In bluegill, 96-hour LC_{50}'s are lower (cyanide is more toxic) at 30°C (0.13-0.14 mg/l as CN) than at 18° (0.17-0.18 mg/l) in both soft and hard water. On the other hand, some evidence indicates that lower concentrations of cyanide ($<$0.3 mg/l as CN) are lethal more rapidly at lower temperatures (Great Britain 1953); and, for rainbow trout, LC_{50}'s are lower at very low temperatures -- between 2-4°C -- (Great Britain 1968, 1972) than at temperatures of 12-20°C.

These differences may be due to slower metabolism at lower temperatures, which would slow down detoxification in the body so that cyanide would be more toxic at moderate concentrations. High concentrations of cyanide, however, may prevent the detoxification process from handling the cyanide load. In this case, toxicity would increase with the rate of metabolism (e.g., respiration rate) (Doudoroff 1976). More research is needed before cyanide's behavior in varying water temperatures of natural environments can be understood.

Reports of the effect of water hardness on freshwater toxicity have been contradictory. Cyanide has been found to be less toxic in hard waters (Henderson _et al_. 1961), more toxic in hard waters (Leclerc and Devlaminck 1950), and unaffected by water hardness (Cairns and Scheier 1963, Burdick _et al_. 1958). Among the studies, the ranges in experimental procedure and hardness differed. This may account for the conflicting results.

One study on salinity and cyanide toxicity reported that the three-spine stickleback, an estuarine fish, died significantly sooner after exposure to 0.27 mg/l and 0.21 mg/l cyanide in seawater (30 ppt salinity) than in diluted seawater (half-strength) and in freshwater (Broderius 1973). No other information was available on estuarine or marine species; consequently, it is difficult to assess whether cyanide is consistently more toxic at high salinities.

Cyanide is more toxic to fish in water containing low concentrations of dissolved oxygen (<4 mg/l). Both the time of survival (Downing 1954, Burdick *et al*. 1958) and LC_{50}'s (Burdick *et al*. 1958, Cairns and Scheier 1958) are reduced at lower oxygen levels.

5.1.3 Toxicity to Wildlife

No data were available concerning the effects of cyanide on wildlife species; therefore, laboratory animal data were examined to provide an indication of the cyanide concentration ranges toxic to small mammals. Table 5-7 presents the results of reported laboratory studies on cyanide toxicity through inhalation and Table 5-8 presents lethal concentrations through ingestion.

When inhaled, cyanide is rapidly lethal to rats and rabbits at concentrations of 100->1000 mg/l. Of those compounds reported, cyanide in the form of HCN is most toxic because of its rapid release to the body.

Ingested cyanide is reported lethal to rats and rabbits at concentrations of 8.7-39 mg/kg body weight. No exposure time was reported; however, cyanide at these concentrations was probably toxic immediately. No studies of long-term exposure to low concentrations of cyanide were available. However, some evidence indicates that mammals rapidly metabolize cyanide, and are not affected by levels too low to be acutely toxic (see Section 6.1).

No information was available regarding toxicity of cyanide to birds, via any exposure route (ORNL/U.S. EPA 1978).

5.2 BIOTIC EXPOSURE TO CYANIDE

5.2.1 Introduction

This section describes the potential for exposure of selected aquatic organisms to harmful levels of cyanide in the ambient environment. The approach used in developing this section was as follows:

The findings of the toxicity assessment for biota (see Section 5.1) were reviewed in conjunction with the U.S. EPA data on reported fish kills attributed to cyanide for the 1972-77 period. This review provided a basis for identifying potentially sensitive and important receptors, and for identifying cyanide levels known or suspected to be potentially harmful in the environment. Certain data were emphasized in reviewing the laboratory data discussed in Section 5.1, i.e., cyanide levels associated with organism mortality under acute or chronic conditions, and cyanide levels associated with reduced mobility or reproductive efficiency. Then, the distribution of selected sensitive/important organisms at the national scale was characterized. This distribution of sensitive receptors was compared

TABLE 5-7. REPORTED EFFECTS OF INHALED CYANIDE AND CYANIDE COMPOUNDS ON LABORATORY ANIMALS[a]

Concentration in air (ppm)	Species	Compound	Response	Source
110 (0.12 mg/1)	Rat	HCN	Fatal in 1.5 hr	Dudley *et al.* (1942)
3.5 (0.35 mg/1)	Rabbit	HCN	Fatal	"
400 (0.85 mg/1)	Rat	Cyanogen	Fatal in 1 hr	McNerney and Schrenk (1960)
400 (0.84 mg/1)	Rabbit	Cyanogen	Fatal in 1.8 hr	Fassett (1963)
1.4 mg/1	Rat	Cyanogen Chloride	Fatal in 0.17 hr	Spector (1956)
1200 (3.0 mg/1)	Rabbit	"	Fatal in 2 min	Fassett (1963)
2.2 mg/1	Goat	"	Fatal in 7-10 min	Spector (1956)

TABLE 5-8. ACUTE TOXICITY OF ORAL-ADMINISTERED CYANIDE TO LABORATORY ANIMALS

Concentration mg/kg of body wt	Species	Compound	Response	Source
8.7-11.5	Rat (male)	Potassium Cyanide	Lethal[a]	Gaines (1969)
15 (11-21)	Rat	Sodium Cyanide	LD_{50}	Smyth *et al.* (1969)
23.5	Rabbit	Cyanogen Iodide	Lethal	Hunt (1923)
39 (30-51)	Rat	Calcium Cyanide	LD_{50}	Smyth *et al.* (1969)

[a] No exposure time reported

with the available information concerning the levels of cyanide found nationally (Chapters 3.0 and 4.0) in order to identify possible areas where aquatic biota may be at risk.

5.2.2 Effects Levels

In assessing aquatic exposure, the initial step was to review the aquatic effects discussed in Section 5.1 in relation to the distribution of cyanide in surface waters.

Effects data were reviewed to identify sensitive, important aquatic organisms for exposure/risk considerations and to identify ambient threshold cyanide concentrations at (or above) levels that would incur adverse effects.

From the laboratory results cited in Section 5.1 and in Towhill *et al*. (1978), the following principal observations can be made:

- The lowest reported levels of cyanide effects were approximately 10 μg/l for both acute and chronic effects. Neill (1957), Broderius (1970), and Leduc (1978) reported, respectively, a 75% reduction in the swimming ability over a one-month period, greater than a 50% reduction in swimming ability in a two-hour period, and damage to developing embryos in greater than a 100-day period for various salmonids exposed to concentrations of cyanide as low as 10 μg/l. Conversations with the U.S. EPA Environmental Research Laboratory staff at Duluth (Personal Communications, Environmental Research Laboratory, Duluth, MN, 1980) indicate that the lowest reported effects level for freshwater fish is a chronic value of 5 μg/l, reportedly associated with an absence of spawning in bluegill sunfish.

- These cyanide levels (∿10μg/l) correspond with a detection limit commonly employed in past analyses of ambient water quality. This implies that much of the available monitoring data (see Section 3.3) may not be sufficiently precise to allow useful projections of risk to the most sensitive receptors.

- The laboratory data summarized in Section 5.1 suggest strongly that the effects of cyanide depend highly on the form of cyanide present in the environment. Since available monitoring data sometimes report only total cyanides, aquatic exposure estimates based on these data are difficult to compare with the dissolved concentrations reported in toxicity studies.

- Little, if any, basis exists for distinguishing unusually sensitive species for the assessment of cyanide risks in the aquatic environment. Most, but not all, of the older laboratory-based data suggest that salmonids were the most sensitive species, exhibiting adverse effects at levels of exposures as low as 10-50 µg/l. However, as previously mentioned, recent work with warm-water species, including bluegills and fathead minnows, suggests that these species can also experience adverse effects as a result of chronic exposure to cyanide concentrations < 50 µg/l.

Based on receptor distribution, no geographic region can be distinguished as a significant area of exposure because these species are widespread in cold and warm waters.

5.2.3 Exposure Levels

The information presented previously concerning sources of cyanide releases to water (see Chapter 3.0), major environmental pathways that determine the environmental distribution of cyanide (see Section 4.2), and cyanide levels actually detected in surface water (see Section 4.3) have the following implications for aquatic exposure levels.

- The fate calculations (see Section 4.2) imply that exposure to cyanide concentrations >50 µg/l are possible in ambient waters receiving discharges from any of the large point-source wastewater dischargers, such as iron and steel facilities, steam-electric power plants, and POTWs. These calculations also imply that such elevated concentrations would be restricted to relatively localized extensions of the receiving waters associated with the several types of discharges. However, considering the limitations of the "uniform mixing" assumption used in these calculations, it is possible that even smaller volumes of receiving water may actually be affected; but that concentrations of cyanide might be higher than the calculated levels.

- Maximum concentrations of cyanide in surface water occasionally exceed the 10-100 µg/l acute effects thresholds discussed earlier. The STORET 85th percentile values (see Section 4.3) suggest that these high concentrations occur infrequently. Some of the available monitoring data report corresponding measurements of the more toxic, free cyanide concentration at given locations. Thus any conclusions regarding exposure based on data for total cyanide alone must be qualified.

5.2.4 Summary of Exposure to Freshwater Organisms

The information presented in this section suggests that the incidence of localized exposure of finfish to harmful concentrations of cyanide may be widespread and may often be associated with one or more types of point source discharges. Because the actual exposure in any specific water body depends on factors such as discharge volume, discharge control, and the nature of receiving water mixing opportunities, no national average relevant to exposure levels exist. Therefore, information is required on individual discharges and compliance with discharge controls for an accurate assessment of exposure levels in areas with point source discharges of cyanide. Even though these data are not available, certain qualitative conclusions concerning aquatic exposure are possible. Where cyanide levels exceed 5-7 µg/l, these concentrations will probably be in the vicinity of point discharge sources, and they will rapidly diminish at increasing distances from the source. Hence, fish populations in surface waters are probably exposed to these levels only at distances of several kilometers rather than within a broad geographic area, unless there are numerous discharge sources clustered together.

5.2.5 Summary of Potential Exposure to Marine Organisms

The limited data available indicate that marine species, such as the pinfish (*Lagodon rhomoboides*) and eels, can be adversely affected by levels of free cyanide between 50 µg/l and 300 µg/l (Daugherty and Garrett 1951, Costa 1965a). Though this range is comparable with the effects ranges reported for many freshwater fish species (see Section 5.1), these data do not provide information on a large enough number of species to be considered fully representative of potentially important marine exposure situations. Marine finfish, such as the Atlantic silverside (*Menidia menidia*), juvenile striped bass (*Morone sexatilis*), and menhaden (*Brevoortia*), have been reported to be sensitive to various other contaminants in laboratory situations; and it is considered reasonably likely that one or more such species could be more sensitive to cyanide than *Lagodon*.

However, in the absence of monitoring data for marine/estuarine waters, the exposure of marine organisms to potentially toxic levels of cyanide cannot be quantified. Reported fish kills (see Chapter 7.0) provide anecdotal evidence of a few instances of high concentrations.

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6.0 EFFECTS AND EXPOSURE -- HUMANS

6.1 HUMAN TOXICITY

6.1.1 Introduction

Cyanide is among the most potent and rapidly acting of all known poisons. The toxic effects of hydrocyanic acid (HCN), sodium cyanide (NaCN), potassium cyanide (KCN), and other soluble inorganic salts are primarily attributable to the cyanide ion (CN) and its tendency for complexation with certain metal ions. Cyanide binding to metallic cofactors has been shown to inhibit 42 enzyme systems (Dixon and Webb 1958). Cytochrome oxidase, a key respiratory enzyme, is especially sensitive to cyanide. A concentration of 3.3×10^{-8} moles/ml of cyanide completely inhibits cytochrome oxidase, thus preventing tissue utilization of available oxygen (Chen and Rose 1952) and causing cytotoxic anoxia. If untreated, it can be fatal, probably as a result of cerebral anoxia (DiPalma 1971).

Due to cyanide's acute lethality, few long-term mammalian studies are available for anlaysis. In addition, human populations are generally not exposed to acutely toxic levels of cyanide (see Section 6.2), except as an occupational hazard; therefore, determinations of acute toxicity in laboratory animals do not appear relevant to human risk. Also, studies of changes in biochemical parameters or transient physiological responses lack evidence of associated mammalian toxicity. Epidemiologic studies that were found are of little use in risk assessment because they do not relate dose to effect; and those values for cyanide bioaccumulation found in the literature do not provide a clear basis for the determination of human exposure. Thus, the available toxicologic data on cyanide presented in this chapter are sufficient for a qualitative analysis of human risk, but are insufficient for a quantitative assessment of the human risk associated with pollutant sources.

6.1.2 Metabolism and Bioaccumulation

For humans, the exposure routes of cyanide are inhalation, ingestion, or absorption through the skin. Regardless of the intake route, cell membranes are highly permeable to free cyanide, resulting in its rapid absorption through alveolar membrane (Polson and Tattersall 1969), intestinal mucosa (Gettler and Baine 1938), or skin (Potter 1950). The percentage of a given dose absorbed is a function of the size of the dose and the absorption rate. Once absorbed, cyanide appears rapidly in the blood and is subsequently distributed throughout the body.

The metabolism of cyanide as summarized by Williams (1959) is shown in Figure 6-1. In most mammalian species, conversion of the cyanide ion to the relatively non-toxic thiocyanate ion creates a major detoxification pathway for hydrogen cyanide and cyanide salts. Organic cyanides also form thiocyanates if they are converted *in vivo* to the cyanide ion

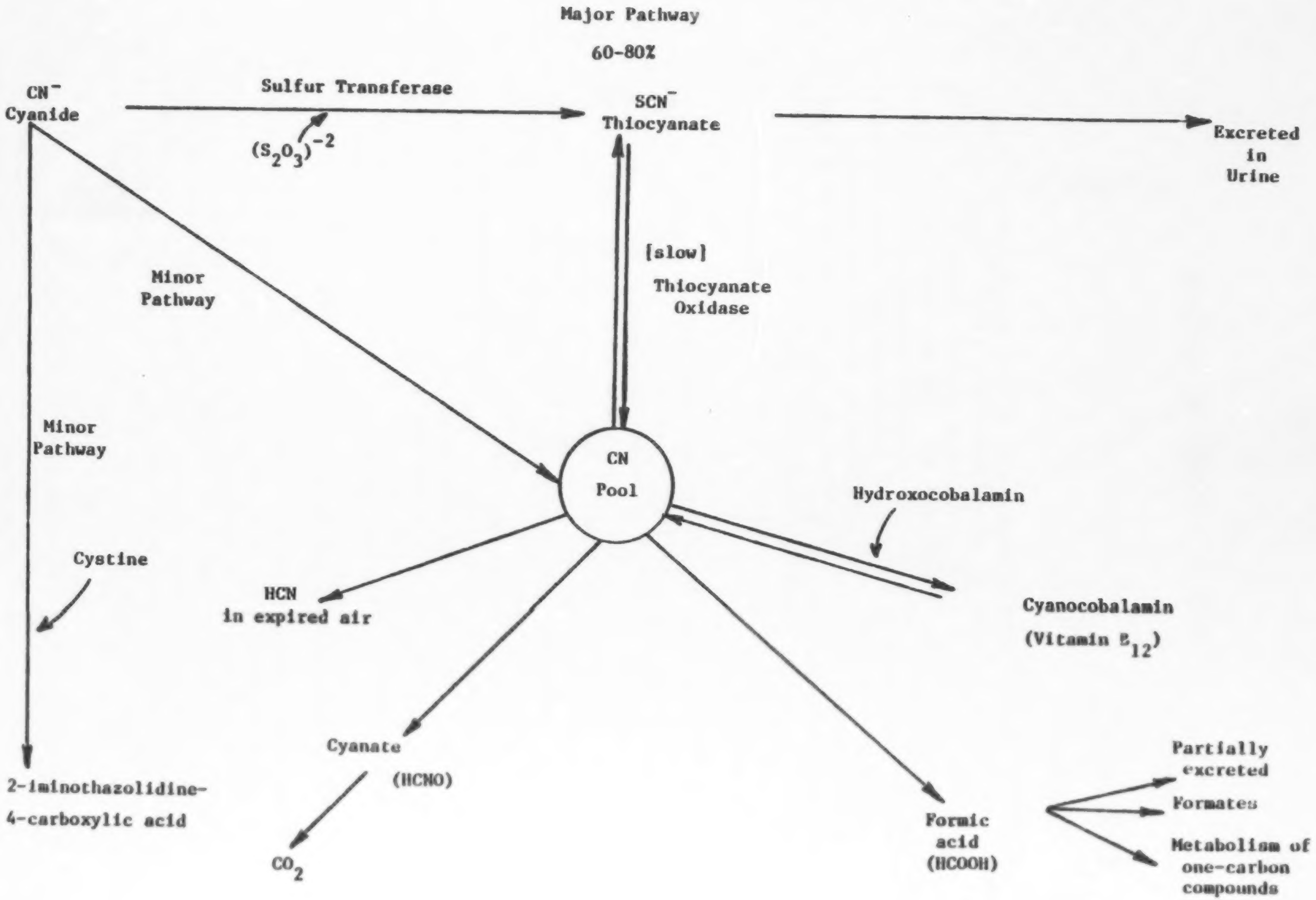
Major Pathway
60-80%
CN⁻
Cyanide
Sulfur Transferase
$(S_2O_3)^{-2}$
SCN⁻
Thiocyanate
Excreted
in
Urine
[slow]
Thiocyanate
Oxidase
Minor
Pathway
Minor
Pathway
CN
Pool
Hydroxocobalamin
Cyanocobalamin
(Vitamin B_{12})
Cystine
HCN
in expired air
Cyanate
(HCNO)
CO_2
2-iminothazolidine-
4-carboxylic acid
Formic
acid
(HCOOH)
Partially
excreted
Formates
Metabolism of
one-carbon
compounds

FIGURE 6-1. Metabolism of Free Cyanide in Mammalian Species

Source: Williams (1959)

(Williams 1959). Rhodanese, the mitochondrial enzyme sulfur transferase that is widely distributed in animal tissues and particularly the liver, mediates this reaction. Rhodanese transfers sulfur from endogenous supplies of thiosulfate, a sulfur donor, to the cyanide ion (CN^-). This forms thiocyanate (SCN^-), which is readily excreted, primarily in the urine. The endogeneous supply of thiosulfate is the step in the detoxification pathway that limits the absorption rate (Williams 1959). A limited amount of thiocyanate can be reconverted to cyanide when thiocyanate is present; however, it will occur at a rather slow rate (Goldstein and Reiders 1953, Himwich and Saunders 1948). Also, the presence of glutathione s-transferases will catalyze a minor conversion of organic thiocyanate to cyanide (Habig et al. 1975).

The conversion to thiocyanate accounts for the detoxification of 60-80% of absorbed cyanide (Williams 1959). Other relatively minor pathways for detoxification and excretion include:

- Combination of cyanide with cystine to form 2-imino-thiazolidine-4-carboxylic acid,
- Oxidation of cyanide to formic acid and carbon dioxide,
- Formation of cyanocobalamin (vitamin B_{12}), and
- Excretion of HCN through the lungs (Williams 1959).

The binding of free CN by methemoglobin in blood also reduces free cyanide levels (Chen and Rose 1952, Williams 1959). McNamara (1976) has estimated the detoxification rate in humans of intravenously administered HCN as approximately 0.017 mg/kg/minute.

Crawley and Goddard (1977) studied the metabolism of $K^{14}CN$ in female rats after intravenous injection, pulmonary and gastric incubation, and skin absorption. After intravenous injection, 4% of the administered radioactivity was excreted in breath, 4% in feces, and 45% in urine within 24 hours; corresponding values after 7 days were 8% for breath, 14% for feces and 68% for urine. The pattern of excretion and levels of radioactivity in tissue found after inhalation exposure were similar to the post-intravenous injection pattern. Some differences in tissue distribution, however, were apparent after skin absorption. Although the evaporation of $K^{14}CN$ from the skin was largely overcome by spraying the area with an artificial skin, some loss (15-30%) by this route still occurred. Approximately 65% of the applied radioactivity was absorbed. At 24 hours, 26% of the absorbed activity was excreted in urine and 7.5% in feces. After 24 hours, residues in fat were higher following skin application than after intravenous injection (7.1% vs. 1.3%, respectively).

After subcutaneous injection of NaCN to rats, 2-amino-4-thiozolidine carboxylic acid could be isolated from urine (Baumeister et al. 1975, Wood and Cooley 1965).

Mehta and McGinity (1977) found no significant difference in the urinary excretion pattern in rats administered 5 mg/kg KCN subcutaneously once or twice a week for 8 weeks. Doubling the dosing rate of KCN resulted in significantly higher levels of urinary thiocyanates; i.e., 1008, 2208, and 126 μg/24 hours for groups receiving one injection/week, two injections/week, and controls, respectively. This suggests that a substrate saturation phenomenon is not operating between these levels of KCN intake. Smith and Foulkes (1966) found that excretion of thiocyanate in the urine of rats administered 1 mg/KCN/rat/week subcutaneously for 20 weeks gradually decreased by 70%. The discrepancy in these two studies may have resulted because Smith and Foulkes (1966) measured thiocyanate concentration in the urine rather than the amount of thiocyanate excreted. Variability in the volume of urine samples could result in highly variable thiocyanate concentrations.

Normal blood cyanide levels are around 0.05 μg/ml in the general population (Feldstein and Klendshaj 1954, Symington _et al_. 1978). Tobacco smoking and/or the ingestion of cyanogenic foods, such as cabbate, spinach, lima beans, etc., are probably the most significant daily sources of cyanide exposure for the general population (Baumeister _et al_. 1975). Because of cyanide's rapid detoxification and excretion, blood cyanide levels are not significantly higher in smokers than in non-smokers. Serum thiocyanate, as well as thiocyanate levels in other body fluids, are elevated in smokers and are roughly proportional to the level of smoking (Maliszweski and Bass 1955). Serum thiocyanate levels typically range from 3 μg/ml in non-smokers to 7 μg/ml in heavy smokers (Levine and Radford 1978). The serum half-life of thiocyanate in humans is 4-8 days (Levine and Radford 1978).

The distribution of cyanide in tissues of cyanide fatality victims indicates that the highest tissue cyanide residues are found in the spleen (78 μg/g), probably resulting from the strong binding of cyanide by erythrocytes; the lowest tissue levels are usually found in brain tissue (5 μg/g) (Sunshine and Finkle 1964). Intermediate concentrations are found in liver, kidney, and blood. The cyanide level of urine if often no greater than normal values because of the rapid lethality of cyanide.

Blood cyanide levels in victims of fatal cyanide poisoning are not entirely reliable in that delays in analyzing blood for cyanide may produce artifacts because of _in vitro_ formation of cyanide or the conversion of cyanide to other products (Ballantyne 1976). For example, studies have shown that to reduce the conversion of cyanide, blood with a low cyanide content should be stored at 4°C, while blood with a high cyanide content is best retained at -20°C (Ballantyne 1976).

Bogusz (1976) also points out that in acute cyanide poisoning with prolonged survival, the application of Co-EDTA causes a rapid decrease in the cyanide level in blood and tissues.

6.1.3 Animal Studies

6.1.3.1 Mechanism of Action

Cyanide has a tendency to form complexes with several metal ions, particularly iron in the ferric (Fe^{3+}) state. Thus, enzyme systems requiring metallic cofactors are susceptible to inhibition via cyanide complexation to these metallic cofactors. Cytochrome oxidase, a terminal enzyme in the mitochondrial electron transport chain, is particularly sensitive to the effects of cyanide; a cyanide concentration of 3.3×10^{-8} moles/ml completely inhibits this enzyme by forming a relatively stable, inactive coordination complex with its ferric ion (Chen and Rose 1952). This complexation blocks utilization of cellular oxygen, producing histotoxic hypoxia. Under ordinary circumstances, the body is capable of handling a small but continuous amount of cyanide by converting to thiocyanate and restoring cell respiration. With prolonged or high exposure levels, however, normal metabolic processes are saturated and cytotoxic anoxia results. Chemoreceptors in the carotid and aortic bodies trigger an inspiratory gasp and hyperpnea (an increase in the depth of respiration). This is followed by a transient depression in the central nervous system and finally hypoxic convulsions and death resulting from respiratory arrest (Fassett 1963, Gosselin et al. 1976).

6.1.3.2 Carcinogenicity, Mutagenicity, and Adverse Reproductive Effects

No definitive studies on the carcinogenic, mutagenic, or teratogenic/reproductive effects of cyanide have been reported. Rats fed a diet fumigated with 300 μg/l HCN exhibited no indications of any carcinogenic effect after 2 years (Howard and Hanzal 1955); however, data are insufficient for definitive conclusions (see Section 6.1.3.3).

Thiocyanate, the major metabolic product of cyanide detoxification, has been shown to produce inhibitory effects at high concentrations (0.7 ml of 1 M NaSCN/egg) on mesodermal and endodermal development of the chick embryo (Nowiniski and Pandra 1946). In view of the high dose and the large number of false positives generated in this closed system, little confidence can be placed on this finding. Furthermore, in a feeding study conducted with pregnant rats, Kreutler et al. (1978) reported no indications of adverse effects in pups born to dams administered 160 μg SCN/ml in their drinking water (∿6.4 mg SCN/rat/day) beginning on day 2 of pregnancy. Although this study was not conducted according to normal teratogenicity testing protocols, it does suggest that a high plasma concentration of thiocyanate is not of itself detrimental to progeny, at least in rats.

6.1.3.3 Chronic Effects

Only one chronic feeding study has been conducted with cyanides. Howard and Hanzal (1955) fed groups of 10 male and 10 female weanling Carworth Farms rats a diet containing an average of 0, 100, or 300 μg/l HCN for 2 years. In order to maintain cyanide concentrations at these levels, fresh food was fumigated with HCN and analyzed every 2 days throughout the study. An average of all food residue analyzed over the 2-year period, however, indicated that the 300 μg/l HCN dropped to 80.1 μg/l, and the 100 μg/l HCN dropped to 51.9 μg/l after 2 days of feeding. Therefore, dietary concentrations are more appropriately expressed as ranges rather than definitive values.

No signs of toxicity were noted during the 2-year study. Food consumption, growth rates, and survival of treated rats were comparable with control animals. At termination, hematologic parameters (unspecified) and gross pathologic findings were within normal limits and compatible with those typically found in aging animals. Histopathologic examination of a representative number of rats (number unspecified) suggested no abnormalities in the heart, lung, liver, kidney, spleen, adrenals, thyroid, testes or uterus and ovary, the cerebrum and cerebellum, and/or stomach. At termination of the study, elevated thiocyanate levels were noted in plasma, red blood cells, liver and kidney of HCN-treated rats.

In view of the limited data and the uncertainties concerning exact dosage, the only conclusion that can be drawn is that 80-300 μg/l HCN in the diet apparently presents no hazard to rats.

6.1.3.4 Subchronic Effects

The available data on subchronic administration of cyanide to experimental animals are also limited. Kreutler and coworkers (1978) examined the effect of prolonged ingestion of potassium cyanide on thyroid weight and thyroid-stimulating hormone (TSH). Young male albino rats were fed a low-iodine, semi-purified diet containing either 2 or 20% casein for 2 weeks. After 2 weeks, these diets were supplemented with 0.2% KCN for an additional 2-week period. A third group of animals received a 2% casein, 0.2% KCN diet that had been supplemented with potassium iodide. The addition of cyanide to a 2% casein, low-iodine diet resulted in significant ($p < 0.05$) increases in thyroid weight and in plasma TSH (see Table 6-1). The effect of cyanide was eliminated, however, by the addition of iodine to the low-protein diet. No effect was noted on thyroid weight, and little or no effect on TSH in rats fed the 20% casein diet supplemented with KCN.

Two additional experiments examined the effects of long-term overloading of thiocyanate, the major breakdown product of cyanide (Kreutler et al. 1978, Ermans et al. 1972). Ermans and coworkers (1972) fed groups of Wistar rats either a low-iodine or iodine-supplemented diet for 4 weeks. Each of the basic diets was further supplemented with 0, 1, 2, or 5 mg thiocyanate per day. In the iodine-supplemented rats, a daily dose of 1 mg SCN/day or greater produced a progressive depletion of the iodine content of the thyroid; the level of plasma $PB^{127}I$ (protein-bound iodine) remained unchanged (see Table 6-2). In the iodine-deficient rats, the iodine content of the thyroid was already reduced by a factor of 10, because SCN was not administered. Chronic overloading with SCN caused

TABLE 6-1. EFFECT OF PROLONGED INGESTION OF KCN ON THYROID WEIGHT AND PLASMA LEVELS OF THYROID-STIMULATING HORMONE IN PROTEIN-DEFICIENT RATS

Diet	No. of Rats	% Change in Body Weight ± SEM	Thyroid Wt. (mg ± SEM)	Plasma TSH (m Unit/100 ml ± SEM)
2% casein	24	- 30 ± 1	8.1 ± 0.6	5.5 ± 1.1
2% casein + KCN	16	- 31 ± 1	17.5 ± 0.7[a]	147 ± 15[1]
2% casein + KCN + KI	14	- 33 ± 1	5.4 ± 0.3[b]	6.3 ± 2.0
20% casein	11	+ 37 ± 3	11.6 ± 0.8	12.5 ± 0.7
20% casein + KCN	10	+ 41 ± 3	10.3 ± 0.5	11 ± 2

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[a] Different from 2% casein group, $p < 0.05$.

[b] Different from 2% casein + KCN group, $p < 0.05$.

Source: Kreutler *et al.* (1978).

TABLE 6-2. ACTION OF LONG-TERM INTAKE OF SCN^- ON THE THYROID SIZE AND THE ORGANIC IODINE METABOLISM IN RATS

Effect	Iodine Supply	Controls	SCN 1 mg/d	SCN 2 mg/d	SCN 5 mg/d
Thyroid Weight (mg/100g)	5 μg/d	10.7	12.7[a]	12.8	11.9
	None	13.4	11.2	14.7	--
Thyroid Iodine content (μg)	5 μg/d	11.9	10.5	7.9[b]	7.6[b]
	None	1.0	0.7	0.6[a]	--
Plasma $PB^{127}I$ (μg/100 ml)	5 μg/d	2.6	2.3	2.2	2.3
	None	1.8	1.3[a]	1.2[b]	--

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[a] Value statistically different from the control value: $p < 0.01$.

[b] Value statistically different from the control value: $p < 0.001$.

Source: Ermans et al. (1972).

an even greater reduction of the iodine content. All of the deficient animals showed marked hyperplasia of the thyroid, but chronic overloading with thiocyanate did not increase the hyperplasia. On the other hand, it reduced significantly the level of $PB^{127}I$ in the serum. The authors suggest that prolonged ingestion of SCN may cause a transitory inhibition of the thyroidal iodide pump, and consequently lead to the depletion of iodine reserves. Another suggestion was the possibility that the increase in renal clearance of iodine resulted from the saturation of tubular reabsorption by thiocyanate.

In another experiment, Kreutler et al. (1978) examined the relationship between ingested SCN, plasma SCN, and thyroid weights in gravid rats and their progeny. Maternal rats were fed either standard laboratory chow or a low-iodine diet. Sodium thiocyanate was added to their drinking water to provide concentrations of 0, 40, 80, or 160 μg SCN^-/ml. The rats drank ~40 ml of water daily; i.e., 0, 1.6, 3.2, or 6.4 mg SCN/rat/day.

Plasma SCN levels in maternal rats were elevated relative to the concentration in their drinking water but varied widely over a fivefold range (251 vs. 1526 μg/100 ml for the 0 and 160 μg/ml groups, respectively, 5 days post partum). Their pups also had increasing plasma SCN levels, but with less variation (146 vs. 279 μg/100 ml, respectively). The addition of SCN to the drinking water of iodine-deficient gravid rats resulted in goiters in both the mothers and their progeny, particularly at the highest concentration level of SCN intake (see Table 6-3). The pups showed a progressive increase in relative thyroid weight as the maternal SCN^- intakes increased.

In summary, thiocyanate resulting from the detoxification of CN, rather than the CN ion directly appears to exert an anti-thyroid action in rats. Thiocyanate is known to compete with iodide for uptake by the thyroid gland (Barker et al. 1941, Ermans et al. 1972); this finding was substantiated by the more pronounced effects observed in iodine-deficient animals.

Three additional studies have examined the subchronic effects of cyanide administered by injection (Gallagher et al. 1975, Smith et al. 1963, Ibrahim 1963). Intraperitoneal administration of increasing daily doses of NaCN (0.1 mg NaCN/kg every second day; dose range 2.5-4.0 mg/kg) to male Wistar rats for 5 weeks produced signs of acute poisoning immediately post-injection but no indications of prolonged toxicity except for decreased body weights at termination (192 vs. 218 g for controlled rats). At necropsy, cyanide-treated rats exhibited no gross or microscopic pathology in the liver, kidney, or central nervous system (the only tissues examined). Copper levels in the liver tissue were significantly lower in cyanide-treated rats (11.3 vs. 15.2 μg/g dry weight for controls) but were not below the normal range of this species (Gallagher et al. 1975).

In the second study, however, noticeable cellular changes in the cortex, hippocampus, and cerebellum with neuronal degeneration and cell loss were noted in three adult Wistar rats injected subcutaneously with 0.5 mg KCN/rat once per week for 22 weeks. No ill effects were noted prior to sacrifice (Smith et al. 1963, Smith and Duckett 1965).

TABLE 6-3. EFFECT OF MATERNAL SCN INGESTION ON THYROID WEIGHT IN RATS 5 DAYS POST PARTUM

	SCN^- in Water ($\mu g/ml$)	Thyroid Weight (mg/100g body wt ± SEM)
Mothers	0	10.7 ± 0.7
	40	13.3 ± 0.2 ($p < 0.05$)
	80	13.6 ± 1.2 ($p < 0.05$)
	160	16.7 ± 1.8 ($p < 0.05$)
Pups	0	23.7 ± 1.6
	40	30.0 ± 2.2 ($p < 0.05$)
	80	33.0 ± 6.0
	160	42.8 ± 5.5 ($p < 0.05$)

Source: Kreutler _et al_. (1978)

Necrotic lesions and demyelination were also observed in the brain tissue of rats given five subcutaneous injections of NaCN/week for 3 weeks. The initial dose was 8 mg/kg, with successive increments of 0.5-1.0 mg up to a maximum of 6 mg/rat/day (Ibrahim 1963).

Similar lesions of the central nervous system have also been observed in dogs following repeated exposures by inhalation to 50 mg/m^3 HCN for varying durations (12-30 minutes) at either 2-or 8-day intervals. Vasodilation and hemorrhages were most pronounced in the central gray nuclei, brain stem, bulb and medulla cervicalis. Cellular lesions were manifested by cytologic changes in Purkinje cells of the cerebellum and in the bulbar gray nuclei. The author concluded that the lesions resulted from anoxia caused by the inhibition of cytochrome oxidase (Valade 1952).

It is not clear if these lesions in brain tissue are related directly to cyanide, thiocyanate, or general cytotoxic anoxia. The lesions are similar to those produced by hypoxia (Bass 1968).

6.1.3.5 Acute Effects

Lethality

The acute toxic effects of cyanide result from the cytotoxic hypoxia that it produces, which in turn depends on the rate of absorption and the duration of exposure. The more rapidly tissues absorb a critical concentration of cyanide, the more severe the effects.

Inhalation of HCN leads to the most rapid absorption in tissues producing reactions within seconds and death within minutes (Gosselin *et al.* 1976). Death from ingestion of HCN or any compound releasing CN via digestive processes and/or intestinal microflora may be delayed as long as an hour, depending on the stomach contents and the release rate of cyanide from the ingested compound (Gosselin *et al.* 1976). Toxic amounts of cyanide may also be absorbed through the skin (Goodman and Gilman 1975).

An acute oral LD_{50} (lethal dose to 50% of test animals) value of 3.7 mg/kg in mice has been reported for HCN with toxicity slightly reduced for cyanide salts; i.e., oral LD_{50} values between 5 and 10 mg/kg in rats, rabbits, and dogs (RTECS 1977). Similar values are noted via intraperitoneal or subcutaneous injection (RTECS 1977).

Gettler and Baine (1938) administered oral doses of 20, 50, and 100 mg of KCN (expressed as HCN) to three dogs; they died in 155, 21, and 8 minutes, respectively. An analysis of the stomach contents indicated that the three dogs had absorbed 14.4, 12.0, and 16.6 mg of KCN (expressed as HCN), respectively. The authors calculated the lethal oral absorbed dose for dogs as 1.06-1.40 mg/kg HCN.

Walton and Witherspoon (1926) attempted to quantify the skin absorption of HCN in rabbits and dogs. These experiments minimized HCN by permitting entry through percutaneous absorption only. In one experiment, the shaved abdomens of eight guinea pigs were exposed to the vapor of a 97% aqueous solution of HCN. Within minutes, the animals exhibited rapid respiration, followed by general twitching of muscles, convulsions, and then death. In a second experiment, these authors exposed the shaved abdomen of nine dogs to various concentrations of airborne HCN. Exposure to 15,200 µg/l HCN for 47 minutes was lethal; however, no effects were noted in dogs exposed to 5000 µg/l HCN for 180 minutes. Fairley and coworkers (1934) reported similar results for rabbits and guinea pigs.

McNamara (1976) compiled a table on inhalation data, which indicates that goats, sheep, pigs, monkeys, and guinea pigs have high resistance to the lethal effects of hydrogen cyanide, whereas dogs, mice, rats, and rabbits are relatively more sensitive (see Table 6-4).

Sato (1955) discovered that approximately 20% of mice exposed to 20 µg/l HCN gas died within 4.5 hours and that some mice died after 4 hours exposure to 15 µg/l HCN. Impaired mobility and respiration were noted in mice exposed to 10 µg/l HCN for 2 hours.

Except for one animal that died after an 8-minute exposure of 50 µg/l HCN, sixteen rats exposed to 24-50 µg/l HCN for up to 22 minutes survived (Moss _et al_. 1951).

Lehmann (1903) noted that inhalation of 30-40 mg/m^3 of HCN (27-36 µg/l) did not affect cats after 4-to 5-hour exposures. However, cats exposed to airborne HCN concentrations of 50 mg/m^3 (45 µg/l HCN) for 1.5 hours exhibited respiratory distress, increased salivary flow, vomiting, dilatation of pupils, and convulsions. Most cats died after exposure to HCN at 50-60 mg/m^3 (45-54 µg/l) for 2.5-5 hours.

Haymaker and coworkers (1952) exposed six dogs individually to concentrations of 620, 590, 700, 700, 165, and 690 mg/m^3 HCN for periods of 2.0, 2.0, 1.75, 1.75, 10.0, and 2.0 minutes, respectively. Dogs exposed to the first three concentrations died within 20 hours; the three remaining dogs were killed at 24, 26, and 28 hours, respectively, post-exposure. Necrosis of brain gray matter was noted in some of the dogs; definite alternations of structure were not observed in dogs that died within 21 minutes of exposure but were noted in dogs that died 2.5 or more hours after exposure. Similar lesions, however, have been reported from anoxia alone.

Aside from lethality, four basic categories of effects result from acute exposure to cyanide: hematological, cardiovascular, neurological, and metabolic. Information regarding these effects is summarized in the following sections.

TABLE 6-4. LETHALITY OF HCN INHALED BY EXPERIMENTAL ANIMALS

	LCt_{50} (mg min/m^3) for Various Exposure Times					
Species	0.5 min	1 min	2 min	3 min	10 min	30 min
Dog	800	700		1000		
	800	616				
Mouse	450	750	1348		2300	5425
	566	911	1268	1100	736	4890
Rat	800	1550	2200	1800		
	769	932	2190			
Rabbit	904	850				
		980			3200	
Sheep	1441					
Cat	1474	850	1226			
Monkey	1616	1700				
Pig	1740					
Guinea pig	2500	2100				
	2112					
Goat	1300		2200			
	2354		2170			

Source: McNamara (1976).

Effects on Metabolism

Isom and Way (1976) reported that intraperitoneal administration of 10 mg/kg KCN (a lethal dose) greatly depressed the enzymatic activity of cytochrome oxidase in liver and brain tissues (15 and 18% of controls, respectively) of male Swiss Webster mice.

Schubert and Brill (1968) found that inhibition of liver cytochrome oxidase in mice, rats, and gerbils reached a maximum 5-10 minutes after the intraperitoneal administration of KCN. Depending on the dose, the enzyme activity returned to normal 5-20 minutes after maximal inhibition in mice but required up to 1 hour or more in rats and gerbils.

Inactivation of cytochrome oxidase by cyanide results in a shift of aerobic to anaerobic metabolism accompanied by a marked accumulation of lactate. The concentration of adenosine triphosphate (ATP) and phosphocreatine decreases and adenosine diphosphate (ADP) increases. This modification of normal metabolism may cause the cell to increasingly utilize alternate pathways and/or to induce minor pathways in order to maintain a balanced redox state and energy pool (Isom et al. 1975).

One of the most important alternate pathways utilized during cyanide exposure is the pentose phosphate pathway. Administration of 5 mg/kg of KCN to mice increased catabolism of carbohydrates by the pentose phosphate pathway with a decline in utilization of the Embden-Meyerhoff-Tricarboxylic Acid cycle and glucuronate pathway (Isom et al. 1975).

Significant cyanide poisoning is also invariably associated with lactic acidosis. As oxidative phosphorylation is prevented by cyanide, the rate of glycolysis is markedly increased through the Pasteur effect. This increases lactic acid generation and leads to lactic acidosis (Graham et al. 1977).

Dechatelet and coworkers (1977) have shown that cyanide stimulates both oxygen uptake and hexose monophosphate shunt activity in phagocytizing human polymorphonuclear leukocytes.

Effects on Cardiovascular System

Electrocardiographic abnormalities observed in dogs given lethal doses of cyanide (as the cyanogenic glycoside of laetrile) included a notable decrease in the heart rate (bradycardia) accompanied by sinus irregularity, and eventually, complete suppression of P waves, ventricular tachycardia, fibrillation, and cardiac arrest (Schmidt 1978).

Reflex bradycardia has been demonstrated in dogs anesthetized by 50 µg of cyanide administered directly into the common carotid arteries. Even after selective surgical denervation of the carotid sinus, the same dose of cyanide produced a marked bradycardia. After surgical denervation of the carotid body alone, however, the same dose of cyanide had no effect on the heart rate. Therefore, it was concluded that the bradycardia originated in the carotid body chemoreceptors (Jacobs et al. 1971).

Christel and coworkers (1977) have demonstrated a correlation of heart rate and respiratory rate with plasma cyanide levels in dogs. Within three minutes after the oral administration of 24 mg/kg KCN, the cyanide concentration in the plasma rose to 40 μM, and respiration and heart rate slowed down. Within a short time, respiration resumed and the heart rate returned to normal.

In an abstract, Laube et al. (1966) reported that low doses of NaCN administered to dogs by slow intravenous infusion produced increased cardiac output; marked increased coronary flow and oxygen saturation of venous sinusal blood; decreased myocardial oxygen, lactate, and pyruvate consumption; increased cardiac respiratory quotient, which was demonstrated by an increased carbon dioxide production; and excess myocardial lactate, which suggests the occurrence of myocardial anaerobic metabolism.

Effects on Brain and Central Nervous System

In a series of experiments with rats and monkeys, Brierley and coworkers (1975, 1976, 1977) demonstrated that histotoxic hypoxia resulting from intake of cyanide can lead to damage in the gray and/or white matter of the brain in the presence of secondary effects on respiration and circulation only.

The critical contribution of the secondary effects of cyanide on respiration and circulation in the genesis of brain damage was confirmed in the rat (Brierley et al. 1976). NaCN (0.014 mg/minute/100g) was infused intravenously into rats while the major parameters of respiratory and cardiac function were recorded. Neuropathological examination of the brains from 19 rats indicated typical ischemic cell change in only one animal in which major secondary effects had occurred, particularly on the circulation (Brierley 1976).

Experiments with 11 adult monkeys (*Macaca mulatta*) demonstrated that the cytotoxic hypoxia resulting from cyanide does not cause neuronal damage until the secondary effects on respiration and particularly on circulation are considerable. Intravenous infusion of NaCN did not produce typical ischemia in the brain or any other type of cell change unless major effects on the circulation were present. In most animals, the EEG, ECG, respiratory rate, blood pressure, cerebral venous sinus pressure, end-tidal pCO_2, and body temperature were recorded. Blood gases, pH, lactate, and pyruvate were estimated in arterial and venous sinus blood samples. An initial hyperventilation occurred with tetany in all animals. A rapid rate of cyanide infusion led to apnea. Bradycardia usually precipitated an isoelectric or near-isoelectric EEG, with additional hypotension. Neither epileptic seizures nor their EEG concommitants were observed at any stage. Three animals died of heart failure. Brain damage of the white matter was seen in four animals that survived up to 98 hours. Ischemic neuronal alterations, restricted to the striatum of one animal, were attributed to major circulatory problems (Brierley et al. 1977).

Lessel (1971) injected rats subcutaneously three times per week with increasing doses of NaCN (0.4-1.75 mg/100g). These rats developed demyelinative and necrotic lesions in the corpora callosa (70% of the animals) and in the optic nerve (20% of the animals). The optic neuropathy was bilateral, focal and retrobulbar. All rats with optic neuropathies had marked callosal lesions (Lessell 1971).

6.1.4 Human Studies

6.1.4.1 Overview

Few controlled studies have been conducted with cyanide in humans, and the data that are available deal primarily with acute exposure. If death does not result from acute cyanide exposure, recovery is usually complete and prompt. The literature reports of alleged sequelae in animals to acute cyanide poisoning; most of these deal with lesions in the central nervous system (Smith *et al*. 1963). These lesions, however, are not believed to be direct effects of cyanide, but rather an indirect effect of CN-induced anoxia (Smith *et al*. 1963). Insufficient data are available on the effects of chronic exposure to low levels of cyanide. Epidemiologic studies, however, have linked chronic cyanide exposure to various human neuropathies, such as tobacco amblyopia, Leber's optic atrophy, and Nigerian nutritional neuropathy (Baumeister *et al*. 1975, Smith and Duckett 1965, Ermans *et al*. 1972, Osuntokun *et al*. 1970).

6.1.4.2 Controlled Human Studies

Oral Toxicity

The human lethal dose of ingested HCN is believed to be 50-90 mg; this corresponds to approximately 1 mg/kg for a 70-kg man. The toxicity of the cyanide salts is somewhat lower because of slower absorption, i.e., 200-250 mg or approximately 3 mg/kg for a 70-kg man (Gettler and Baine 1938).

Recoveries, however, from the ingestion of 3-5 g KCN without therapy (Liebowitz and Schwartz 1948) and from 4-6 g KCN with prompt treatment (Isom *et al*. 1975) have been reported. Edwards and Thomas (1978) reported the survival of a 48-year-old chemist who swallowed 413 mg of pure KCN on an empty stomach. When admitted to a hospital, approximately 40 minutes later, the subject was unconscious with unrecordable blood pressure; he underwent cardiorespiratory arrest immediately. Spontaneous respiration returned at three hours with supportive treatment; consciousness returned after 8 hours. Approximately 60 minutes after ingestion, peak blood cyanide reached 38 mg/l. Similar findings were observed in a 21-year-old male who survived ingestion of 600 mg of KCN. In this case, lactic acidosis and pulmonary edema were the major manifestations of cyanide poisoning (Graham *et al*. 1977).

Results of oral CN intoxication, however, must be interpreted carefully as the presence of food in the digestive tract may retard absorption. Gettler and Baine (1938) reported that absorption of 0.5-1.5 mg/kg expressed as HCN is lethal to humans regardless of the amount of cyanide ingested.

Clinical manifestations of cyanide intoxication by ingestion can appear within seconds to minutes of exposure. If the stomach is empty and the free gastric acidity is high, absorption and poisoning are rapid. Toxic symptoms include vertigo, hyperpnea, dyspnea, headache, palpitations, cyanosis, and unconsciousness with asphyxial convulsions, preceding death (DeBusk and Seidl 1969). After ingestion of cyanide salts, however, death may be delayed as long as an hour (Gosselin et al. 1976).

Dermal Toxicity

Diffusion cell measurements of absorption of $NA^{14}CN$ solutions (1, 10, or 40% w/v) across human epidermis (1.8 cm^2) at 30°C indicate that absorption rates are strongly dependent on pH. The permeability constant for HCN was calculated to be 25 times greater than that of cyanide ion (Dugard and Mawdsley 1978).

Skin contact with concentrated cyanide solutions can be lethal (Tovo 1955) or produce permanent disability (Collins and Martland 1908). Contact with inorganic cyanide solutions as dilute as 0.5% KCN have produced headaches, dizziness, and skin irritation (Nolan 1908). The typical skin lesion is manifested in eczematoid dermatitis, rash, or skin discoloration (Tovo 1955, Collins and Martland 1908, Nolan 1908). Prolonged intimate contact with solutions of cyanide salts has caused caustic burns and even death (Tovo 1955).

Cohen and coworkers (1974) reported no nasal or skin irritation in 15 human volunteers who underwent dermal exposure to 0.006 mg/m^3 of airborne cyanide.

Inhalation Toxicity

Although the fatal human inhalation dose of HCN has not been firmly established, it appears that concentrations above 90 µg/1 ($\sim$ 100 mg/m^3) are lethal (Flury and Zernik 1931, Lazareff 1956, Lazareff 1971).

Lethality, however, is a function of both concentration and duration of exposure because exposure to concentrations of 90-135 µg/1 may be fatal after 30-60 minutes, while exposure to 300 µg/1 is fatal within a few minutes (NIOSH 1976).

Controlled experiments with human volunteers have not exceeded 500-625 mg/m^3 for approximately 1-minute durations. Grubbs (1917) reported no ill effects on several volunteers exposed to $\sim$ 501 mg/m^3 NaCN gas for 2 minutes or 750 mg/m^3 for 1-1/2 minutes. Similarly, Katz and Longfellow (1923) reported that in experiments during the war, men were exposed to concentrations of 500 mg/m^3 for approximately 1 minute without injury.

Barcroft (1931) records how he submitted himself (70 kg) and a dog ($\sim$ 12 kg) to an atmosphere of 550-688 mg/m^3 HCN until the dog became unconscious (91 seconds). The dog subsequently recovered and

Barcroft reported no adverse effects except for a momentary feeling of nausea; he experienced difficulty in concentrating on conversation approximately 10 minutes after exposure terminated.

Wexler et al. (1957) observed the execution of four men by HCN inhalation (concentrations were not reported). These men exhibited striking electrocardiographic aberrations and had a marked decrease in heart rate during the first three minutes, accompanied by sinus irregularity and eventually, by the complete disappearance of P waves. A secondary increase in heart rate, but not up to the pre-exposure rate, was observed during the 3rd and 4th minutes along with the irregular reappearance of P waves. All four subjects showed A-V dissociation with a secondary decrease in rates during the 5th minute. During the 6th and 7th minutes, the heart rates again showed a slight increase and a return to normal sinus rhythm. Thereafter, the heart rates decreased progressively. Normal A-V conduction in one man and incomplete A-V block in another were maintained throughout the 13-minute observation. A third subject developed a 2:1 heart block, and, finally, a complete heart block. The fourth subject's heart had normal A-V conduction until the 14th minute, when it developed ventricular tachycardia and ventricular fibrillation. Although these changes in cardiovascular activity may not reflect the effects of lower concentrations of cyanide, they do indicate that cyanide exerts no specific effect on the myocardium, but rather induces effects typical of hypoxia. Another complication of the toxic effect of cyanides on the myocardium is left ventricular failure and increased pulmonary capillary pressure resulting in pulmonary edema (Graham et al. 1977).

Intravenous Administration

Sixteen healthy soldiers were given 0.11-0.2 mg NaCN/kg intravenously to stimulate respiration. Electrocardiograms of 15 of the 16 men revealed a sinus pause, without evidence of auricular acticity, persisting for 0.88-4.2 seconds. This sinus pause immediately preceded or accompanied the respiratory stimulation. Immediately after the pause, marked sinus irregularity and a decreased heart rate occurred, which persisted for periods ranging from a few seconds to 2 minutes. Heart rates then accelerated to above pre-injection rates. Heart rate and rhythm were generally restored within three minutes. The 16th subject failed to show a sinus pause and exhibited only a slight acceleration in heart rate. One of the subjects reported a momemtary "dim-out" during the test (Graham et al. 1977).

6.1.4.3 Epidemiologic Studies

The literature contains many reports of accidental or intentional fatal poisoning by cyanide; however, the dose is frequently unknown (Gettler and Baine 1938, Bogusz 1976, Winek et al. 1978, Braico et al. 1979). Winek and coworkers (1978) reported that blood cyanide levels in six cases of suicide by cyanide ingestion ranged from 0.4 mg% to 4.5 mg%. In addition, NIOSH (1976) reports several cases of accidental occupational exposure to HCN and cyanide salts. Since epidemiologic studies have not generally related dose to effect, and have been reported in detail elsewhere (NIOSH 1976), they will not be reviewed in detail here.

Hardy and coworkers (1950) described 2 men exposed continuously to low concentrations of HCN (4-6 μg/l) aerosols generated by case hardening. Both men complained of headache and fatigue. They demonstrated a slight lid lag, enlarged thyroids, and excessive perspiration. One of the two men also experienced dizziness and mental confusion, slurring of speech, occasional abdominal cramps, nausea, vomiting and a coarse tremor of the extremities followed by a temporary (24-hour) paralysis. The enlarged thyroids are probably attributable to the thiocyanate resulting from continuous detoxification of minute quantities of cyanide.

Of 36 non-smoking male employees exposed to 4.2-12.4 μg/l cyanide in electroplating shops, 20 (56%) had slight to moderate thyroid enlargement. No correlation was found between the period of exposure and the size of the thyroid. Thyroid^{131}I uptakes at 4 and 24 hours were significantly higher ($p < 0.001$) in workers than in 20 normal male controls, (38.7 vs. 22.4% and 49.3 vs. 39.9%, respectively). The 72-hour PB^{131}I was within normal limits. Hematologic studies indicate CN-exposed workers had significantly higher hemoglobin and lymphocytic counts than controls. Workers also reported the following symptoms of exposure: headache, weakness, changes in taste and smell, perception, irritation of the throat, vomiting and effort-dyspnea; lachrymation, abdominal colic, precordial pain, and nervous instability were reported less frequently (El Ghawabi _et al_. 1975).

Chronic intake of cyanide from tobacco smoke and/or ingestion of cyanogenic foods has been implicated as a contributing factor to several human diseases, i.e., tobacco amblyopia, retrobulbar neuritis with pernicious anemia, optic atrophy of Leber, and Nigerian nutritional ataxic neuropathy (Osuntokun _et al_. 1970).

Nutritional ataxic neuropathy is prevalent in Nigeria and has been linked with the intake of cassava, a staple root crop containing cyanogenic glycosides. In Nigerian patients with this disease, elevated plasma and urinary levels of thiocyanate are associated with lesions of the skin, mucous membranes, optic and auditory nerve atrophy, and sensory spinal ataxia (Baumeister _et al_. 1975, Osuntokun _et al_. 1970, Ermans _et al_. 1972).

Osuntokun and coworkers (1969) compared the prevalence of neurologic disorders in two Nigerian villages that differed in the amount of cassava eaten (64.3% cassava meals vs. 10.8% in the other village), but were similar in populations (mean age, weight, height, etc.). Degenerative neuropathy occurred with a relatively high frequency in the village with high cassava consumption. Factors other than cyanide intake, however, may be involved, such as environmental or genetic differences.

Ermans and coworkers (1972) have also suggested that diets high in cassava and low in iodine and protein may contribute to the development of goiter and cretinism. These changes are attributable to SCN^- which competes with iodide for uptake by the thyroid gland. Cyanide intake is also associated with neuropathies, particularly tobacco amblyopia and Leber's optic atrophy, which are characterized by visual field defects (Baumeister _et al_. 1975, Smith and Duckett 1965).

Tobacco amblyopia occurs only in smokers; its etiology is believed to be related to an inability to detoxify cyanide. The main symptom of this disease is a central scotoma (an area of depressed vision within the visual field). Vascular changes and degeneration of ganglion cells in the retina and the nerve fibers of the nervus opticus are also seen (Baumeister *et al*. 1975). Data substantiating B_{12}, particularly hydroxocobalamin, as an effective treatment supports speculation that the detoxification of cyanide is distributed in these patients (Chisholm *et al*. 1967).

Leber's optic atrophy may be a congenital abnormality in cyanide metabolism, which becomes apparent when the body is confronted with a source of cyanide. Patients with this disease have a significantly higher proportion of cyanocobalomine in the total B_{12} content of serum than persons having normal vision (Baumeister *et al*. 1975).

Although several reports have connected cyanide with several neuropathies, the evidence is primarily circumstantial. In addition, they are not epidemiologic data and, therefore, cannot be incorporated into a risk assessment.

6.1.5 Summary

6.1.5.1 Ambient Water Quality Criteria - Human Health

The U.S. EPA (1980) has established an ambient water quality criterion for cyanides of 200 ug/l for the protection of human health from the toxic properties of cyanide ingested through water and contaminated aquatic organisms. The criterion is based on the U.S. Public Health Service Drinking Water Standards of 1962.

6.1.5.2 Other Risk Considerations

Cyanide is an acute poison, which is readily absorbed from the alveolar membrane, intestinal mucosa and/or skin, and rapidly appears in the blood. The more quickly a critical concentration of cyanide is attained in the tissues, the more severe the effects. In sufficient doses, cyanide produces rapid death by inhibiting key respiratory enzymes and thereby preventing the body from utilizing available oxygen. At nonlethal doses, cyanide is detoxified to the relatively nontoxic thiocyanate ion. Thus, exposure to small but continuous doses of cyanide may produce no visible effects, while high doses of cyanide over a short time interval saturate normal detoxification mechanisms, which results in acute lethality. Minimum lethal doses of HCN for humans are approximately 50-90 mg by ingestion and approximately 100-150 mg/m^3 by inhalation.

No indications of adverse effects were noted in the single long-term study available for cyanide; however, limitations of the study do not allow definitive conclusions. No data were available on the carcinogenic, mutagenic, or teratogenic/reproductive effects of cyanide. Subchronic studies suggest that thiocyanate, the major metabolite of cyanide, may exert an antithyroidal action in rats. The majority of available experimental data deal with the acute effects of exposure to levels of cyanide not normally encountered by humans from pollutant sources.

In humans, cyanide exposure occurs through ingestion, inhalation, or skin absorption. Acute exposure to cyanide leads either to death or complete recovery. Little is known about the effects of chronic exposure to low levels of cyanide. Epidemiological studies have circumstantially implicated cyanide exposure as a factor in several neurological disorders. Dose/effect data were insufficient to permit quantification of risk; however, the data do suggest that long-term overloading with thiocyanate resulting from cyanide exposure may be a factor in certain human debilities or diseases.

6.2 HUMAN EXPOSURE TO CYANIDE

6.2.1 Introduction

The preceding sections have demonstrated that while cyanide is used widely, its presence in the environment is generally localized. Thus, significant exposure of the general population to cyanide would be unlikely. However, certain exposure situations do exist that are higher than normal safe levels. This section will attempt to identify those subpopulations exposed to higher levels of cyanide through various routes.

6.2.2 Ingestion

6.2.2.1 Food

The primary source of cyanide in food is cyanogenic glycosides. Plants containing these natural compounds produce HCN upon hydrolysis. As many as 1000 species of plants, including such edible items as almonds, stone fruits, sorghum, cassava, lima beans, sweet potatoes, maize, millet, sugarcane, and bamboo shoots exhibit the capability to produce HCN (Conn 1969, Ermans *et al*. 1972). Through the consumption of these foods, cyanide has contributed to the mortality of both humans and livestock.

Maximum concentrations of cyanide released by bitter cassava, sorghum, and lima beans may be as high as 100-300 mg/100 g (Ermans *et al*. 1972). These high levels are primarily concentrated in areas of Africa where cassava is the basic food for millions of people (Conn 1969). Breeding or choosing low cyanide varieties of some species has decreased the consumption of cyanide (Conn 1969). For example, different varieties of cassava roots may contain from 27-378 mg/kg cyanide (Esquivel and Maravalhas 1973). Depending on the variety, lima beans may contain 10-312 mg/100 g cyanide. Samples of the two U.S. varieties, Arizona and America, were found to contain 17 and 10 mg/100 g, respectively (Montgomery 1969).

Thus, human exposure in the United States to naturally-occurring cyanide in food would generally be low. No estimation of average cyanide intake from this source was performed because of variations in the cyanide content of food. However, a worst-case consumption could be as much as 300 mg/day, assuming a concentration of 300 mg cyanide/100 g, and a consumption of 100 g. Such a dose would probably be lethal, but is not likely to occur in the U.S., where both cyanide concentrations and consumption of such foods is lower.

Exposure to cyanide can result from residues in food. Both HCN and $Ca(CN)_2$ are registered as fumigants of several foods including citrus fruits and grains (Ouelette and King 1977). Tolerances established for these uses are shown in Table 6-5. Towill et al. (1978) report that these residues can persist for an extensive time period. However, no basis was given for this statement. If the tolerance for citrus is 50 mg/kg, assuming 200 g/day consumption, the maximum expected exposure would be 10 mg/day cyanide.

6.2.2.2 Drinking Water

The current recommended limit of cyanide in drinking water is 0.01 mg/l, and a mandatory limit of 0.2 mg/l (U.S. DHEW 1962). In 1970, a survey of 969 water supply systems in the United States was taken to assess the quality of drinking water compared with the 1962 U.S. Public Health Service Drinking Water Standards. The maximum concentration of cyanide found in 2,595 distribution samples was 0.008 mg/l (U.S. DHEW 1970). The U.S. EPA (1975) conducted a survey of water supply systems serving interstate carriers. Of 297 analyses for cyanide, 21 or 7.1% failed to meet the recommended limit. The maximum reported level of cyanide was 0.260 mg/l, although this level appeared somewhat questionable. Using this value as a worst case and assuming a 2-l/day consumption, a maximum expected exposure to cyanide from drinking water would be 0.5 mg/day. A more prevalent exposure level, at concentrations of less than 0.010 mg/l, would be 0.02 mg/day.

6.2.3 Absorption

Dermal exposure to cyanide can occur in an occupational setting as discussed in the next section. Dermal exposure to the general population through water has not been assessed; however, it is expected to be very low, considering the low concentration of cyanide in drinking water.

6.2.4 Inhalation

6.2.4.1 Occupational Exposure

A detailed discussion of occupational exposure to cyanide is beyond the scope of this report. The U.S. DHEW (1976) reported that the number of workers with potential exposure to HCN and NaCN has been estimated at 1,000 and 20,000, respectively. Table 6-6 lists occupations with potential exposure to cyanides. The NIOSH recommended standard for

TABLE 6-5. TOLERANCES ESTABLISHED FOR HCN AND $Ca(CN)_2$ IN FOOD

Food	Tolerance (mg/kg)	
	HCN[a]	$Ca(CN)_2$
Spices	250	N.A.
Barley, buckwheat, corn and other grains	75	25
Citrus fruits	50	N.A.
Almonds, beans (dried), cashews and other nuts	25	N.A.
Cucumbers, lettuce, radishes, and tomatoes	N.A.	5

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[a] Past harvest use

N.A. = not applicable

Source: 40:CFR 180

TABLE 6-6. OCCUPATIONS WITH POTENTIAL EXPOSURE TO CYANIDES

Acid dippers
Acrylate makers
Acrylonitrile makers
Adipic acid makers
Adiponitrile makers
Aircraft workers
Almond flavor makers
Ammonium salt makers
Art printing workers
Blacksmiths
Blast furnace workers
Bone distillers
Bronzers
Browners, gun barrel
Cadmium platers
Case hardeners
Cellulose product treaters
Cement makers
Coal tar distillery workers
Coke oven operators
Cyanide workers
Cyanogen makers
Disinfectant makers
Dyemakers
Electroplaters
Executioners
Exterminators
Fertilizer makers
Firefighters
Fulminate mixers
Fumigant makers
Fumigators of fruit trees, apiaries, soil, ships, railway cars, warehouses, stored foods
Galvanizers
Gas purifiers
Gas workers
Gilders
Gold extractors
Gold refiners
Heat treaters
Hexamethylenediamine makers
Hydrocyanic acid makers
Hydrogen cyanide workers
Insecticide and rodenticide makers
Jewelers
Laboratory technicians
Metal cleaners
Metal polishers
Methacrylate makers
Mirror silverers
Mordanters
Nylon makers
Organic chemical synthesizers
Oxalic acid makers
Phosphoric acid makers
Photoengravers
Photographers
Pigment makers
Plastic workers
Polish makers
Rayon makers
Rubber makers
Silver extractors
Silver refiners
Solderers
Steel carburizers
Steel hardeners
Steel galvinizers
Tannery workers
Temperers
Tree sprayers
White cyanide makers
Zinc platers
Zinkers

Source: U.S. DHEW (1976)

employee exposure to HCN is 5 mg/m^3 CN (4.7 mg/kg) for an 8-hour work period. This level was determined as a ceiling concentration based on a 10-minute sampling period. The recommended standard for exposure to cyanide salts is also 5 mg/m^3 CN, with HCN and cyanide salts not to exceed a combined value of 5 mg/m^3 (U.S. DHEW 1976). The NIOSH criteria document also contains recommendations on medical surveillance, labeling of HCN, and salts, personal protective equipment and clothing, work practices and control procedures, and monitoring and recordkeeping requirements. Thus, it appears that this agency has considered cyanide exposure in the workplace in some detail.

6.2.4.2 Exposure of the General Population

The materials balance in Chapter 3 suggests that automobile exhaust is the largest distributed source of cyanide to the atmosphere. Fate calculations estimated a concentration of 0.065 $\mu g/m^3$ cyanide in the air of the northeastern United States. For an average inhalation rate of 19.2 m^3/day, an exposure of 1.25 μg/day would result.

6.2.4.3 Exposure of Identified Subpopulations

Certain subpopulations have been identified as having potentially higher exposure to cyanide than the general population. These subpopulations include smokers; residents near manufacturing facilities, smelters, etc.; persons exposed to high levels of automobile exhaust; and persons exposed to fires.

Exposure through Cigarette Smoke

The Surgeon General's Report (1979) has stated that HCN in mainstream (inhaled) smoke varies from 10 to 400 μg/cigarette in U.S. commercial cigarettes. Thus, exposure of smokers to cyanide could range from 10 to 40,000 μg/day HCN, depending on the type of cigarette smoked, the amount inhaled, and the number of cigarettes smoked, assuming a range of 1-100 per day cigarettes smoked (Surgeon General 1979). Considering that an estimated 33.2% of adults over 17, or 54.1 million persons smoke in the United States (Surgeon General 1979), this exposure is widespread. Of smokers, 25-30% smoke more than 25 cigarettes/day (Surgeon General 1979); thus, a large segment of the population could be exposed to cyanide levels in the 250 to 10,000 μg/day range, or greater.

In addition, non-smokers may be exposed to cyanide through inhalation of sidestream smoke. The Surgeon General's Report (1979) states that sidestream smoke contains 0.6-37% as much HCN as mainstream smoke, or a maximum of 160 μg/cigarette in the sidestream smoke. Although no measurements of HCN have been taken in smoke-filled rooms, concentrations may be estimated from measurements of CO levels, which have been summarized by Burns (1975). The results are not consistent but apparently depend on a number of variables, such as room size, number of smokers, and ventilation. They show levels of 38-80 μg/l CO in rooms (38-93 m^3) where 30-80 cigarettes had been smoked with no ventilation. The Surgeon General's Report (1979) reported levels of 10 to 20 mg CO in mainstream smoke/cigarette with a sidestream/mainstream CO ratio of 2.5, or a maximum of 50 mg CO produced in sidestream smoke/cigarette. Using the ratio between CO and HCN in sidestream smoke (50 mg CO/160 μg HCN) and an assumed room concentration of 80 mg/kg CO, a level of 0.3 mg/m^3 HCN is calculated. Alternatively, using 160 μg HCN/cigarette and assuming a room size of 48 m^3 with no ventilation in which 40 cigarettes were smoked, a concentration of 0.13 mg/m^3 HCN is calculated. Problems do result from using CO levels to estimate HCN levels because CO in a smoke-filled room would be due to sidestream and exhaled mainstream smoke. In addition, the concentration of CO and HCN would be influenced by the type and amount of tobacco smoked, extent of inhalation, size of room, ventilation, and duration of smoking (Surgeon General 1979). Though actual measurements of HCN in such a situation might be enlightening, the maximum calculated values for exposure are still lower than the occupational standard of 5 mg/m^3 (U.S. DHEW 1976).

Exposure of Residents near Manufacturing Facilities and Other Air Dischargers

The materials balance considerations have shown that cyanide discharges to air do occur. The only monitoring data available come from Sofia and an "industrial area" in Bulgaria (Kalpasanov and Kurchatova 1976) where a maximum concentration of 0.013 mg/m^3 HCN was reported. Using an inhalation of 19.2 m^3/day, an inhalation exposure of 0.25 mg/day was estimated.

Exposure from Automobile Emissions

Previous sections have shown that concentrations of cyanide in urban air largely result from automobile emissions. The calculated 90th percentile, 1-hour average in New York City was 0.031 mg/m^3, while the maximum hourly reading calculated was 0.057 mg/m^3. Using this maximum, inhalation in New York City might be as high as 1.1 mg/day.

Exposure in Fires

Carbon monoxide is a significant toxic substance produced in fires. Hydrogen cyanide and other organic cyanides, however, may be released from burning urethanes, acrylonitriles or polyamides in plastics and may result in some hazard to persons exposed. Symington *et al*. (1978) investigated cyanide exposure in fires through blood cyanide and thiocyanide levels. Statistical analysis of the results by these authors showed that non-fatal and fatal casualties (overcome by smoke) showed significantly elevated cyanide levels. The authors suggested that some of the subjects may have been exposed to lethal cyanide concentrations, but generally the major effect of cyanide in fires is its contributions to the effects of carbon monoxide.

Gold *et al*. (1978) examined the air immediately surrounding firefighters for evidence of cyanide exposure. HCN was detected in about one-half the samples taken. Of the 43 samples in which cyanide was detected, 11 were from fires involving specific materials, such as an upholstered chair, mattresses, tires, vehicles, and rubber insulated wire. The maximum reported level of HCN was 2.8 mg/m^3 in air. Exposure would depend on the time involved. Using the maximum concentration, however, and a maximum three-hour exposure without breathing apparatus, an exposure of 13.4 mg/three-hour period would result. (This time is strictly an example. Firefighters are the only persons who purposely expose themselves to smoke for such extended periods and perhaps longer.)

Levine and Radford (1978) calculated the absorbed cyanide from levels of thiocyanate in firefighters. The maximum cyanide absorbed from fires was calculated to be between 0.24-0.36 mg CN/kg body weight. This estimate is higher than the estimate described above, but the assumptions involved in both cases do not allow a preference. Therefore, a firefighter may be exposed to 0.0003-0.4 mg CN/kg, or 0.02 to 28 mg per exposure.

6.2.5 Summary

Table 6-7 summarizes various estimates of exposure for cyanide. This table demonstrates that smokers are probably receiving the largest amounts of cyanide. Firefighters, who are subject to occupational exposure, also may receive high doses. With the exception of smokers, the general population is not exposed to large amounts of cyanide. Levels in drinking water and ambient air are low. Although levels in some food may be high, they are probably low in the United States. Thus, other than naturally-occurring cyanide, no evidence of significant exposure to cyanide in the human diet exists.

TABLE 6-7. ESTIMATED HUMAN EXPOSURE TO CYANIDE[a]

Population	Size of Population	Route of Exposure	Estimated Exposure (mg/day)
General	Large	Food-ingestion	Worst-case 10-300
General	Large	Drinking water-ingestion	Maximum 0.02
	Small	Drinking water-ingestion	Worst-case 0.5
General-Northeastern-U.S.	Large	Inhalation-ambient air	0.00125
Smokers	Large-54.1 million adults	Inhalation	0.01-40
Non-smokers exposed to smoke-filled room, time factor	Potentially large	Inhalation	2-6
Residents near industrial areas	Small	Inhalation	0.25
Residents in urban areas	Large	Inhalation	1.1
Firefighters or other persons exposed to fires	Small	Inhalation	0.02-28 mg exposure

[a] See text for assumptions.

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7.0 RISK CONSIDERATIONS

7.1 RISK CONSIDERATIONS FOR HUMANS

Based upon the exposure and toxicity estimates in the preceding chapters, the general population of the U.S. does not appear to be under any substantial risk due to the discharge of cyanide into the environment. Levels of human exposure to cyanide are for the most part well below the acute effect thresholds for humans. The inhalation effect threshold is about 100 mg/m^3, whereas through ingestion the threshold is about 1 mg/kg body weight. Isolated instances can occur where the oral threshold is exceeded by ingestion of food containing high concentrations of naturally-occurring cyanides. However, cyanide levels in U.S. drinking water are less than 8 µg/l. Smoke inhalation, especially for cigarette smokers, may result in high doses, but over the time period involved the body's detoxification mechanisms provide adequate protection. The only reasonable scenario under which acute poisoning is possible would be accidental ingestion or inhalation of a large single dose over a short time interval. This could conceivably occur in an occupational setting, or as a result of contact with a major cyanide discharge site. However, the probability of individual exposures of this type is negligible. Risk quantification is not feasible without additional investigation.

The potential exposures of humans to cyanide and the potential acute effects of cyanide inhalation or ingestion are summarized in Figure 7-1. The range of acute lethal doses for both exposure routes is well in excess of the typical environmental exposure levels. Intake through inhalation over a short period (1 hour or less) may be above 10 mg for heavy smokers or people exposed to fires, as shown previously in Table 6-7, but these are the only instances in which inhalation may possibly approach a lethal level. The fatal human inhalation dose of HCN is not firmly established, but concentrations above 100 mg/m^3 are generally fatal to living organisms. In addition, ingestion of large quantities of foods containing naturally occurring cyanide is the only scenario that might conceivably result in a lethal ingested dose. Sublethal doses can be tolerated with no permanent adverse effects, due to the body's detoxification mechanism, so that the risk of acute effects due to ingestion appears to be extremely low.

The chronic or subchronic effects of cyanide exposure are not completely understood, due to the limited availability of toxicologic data. Cyanide has been implicated with thyroid disorders, and various neuropathies, but appears to play only an indirect role in the incidence of such disorders via long-term overloading with thiocyanate, cyanide's major detoxification product. In the absence of suitable dose-response data, no quantitative evaluation of chronic risk can be made. However, it is certainly possible that subpopulations with high chronic exposure levels, such as smokers or firefighters, may be susceptible to increased incidence of these diseases.

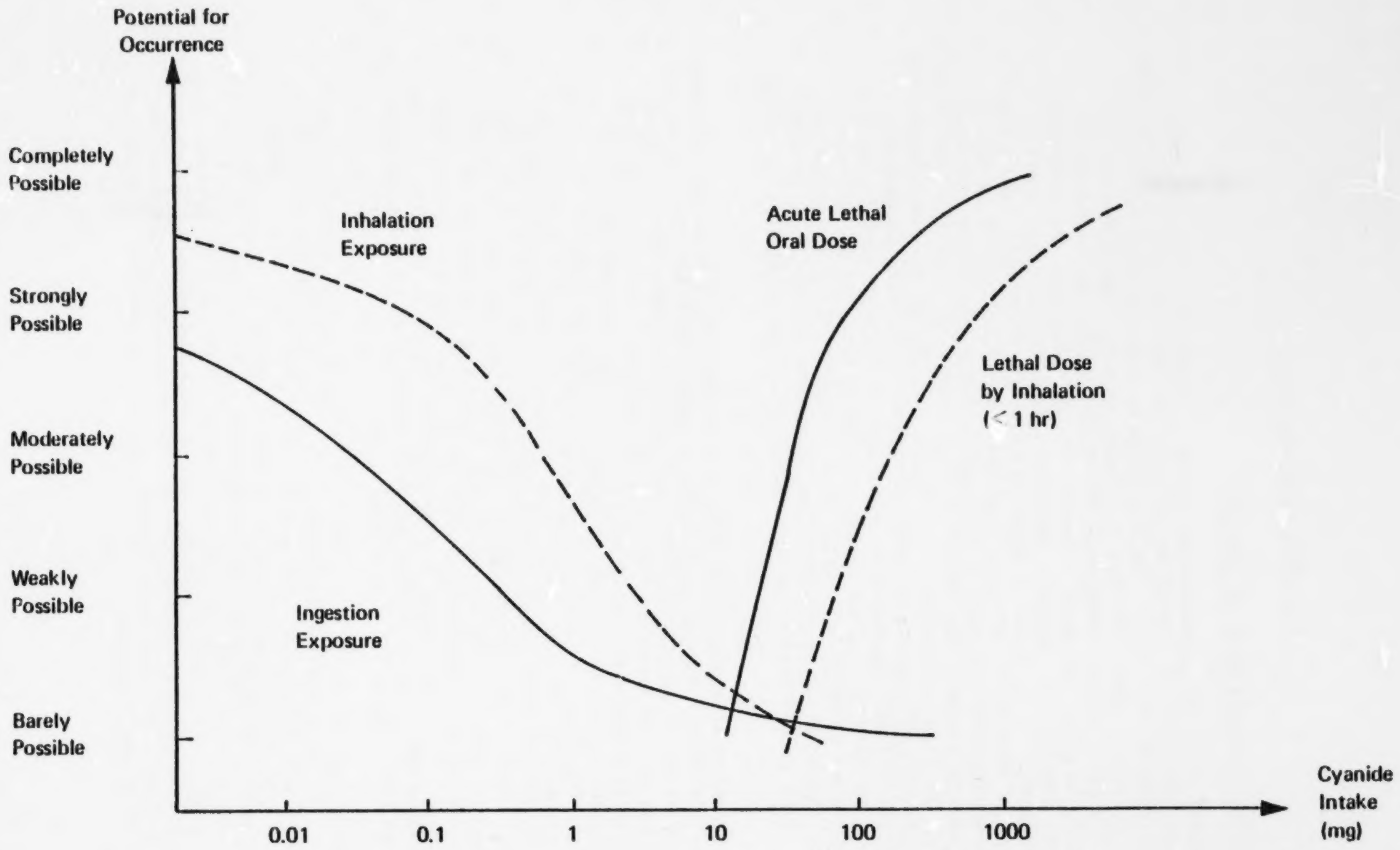

FIGURE 7-1 COMPARISON OF RANGES OF UNCERTAINTY FOR ACUTE EFFECTS OF AND EXPOSURE TO CYANIDE IN HUMANS

7.2 RISK CONSIDERATIONS FOR NON-HUMAN BIOTA

The information presented in this report suggests that the incidence of localized risks to finfish due to exposure to cyanide may be widespread and may often be associated with one or more types of point-source discharges, particularly those associated with iron and steel manufacture, steam-electric power plants, and electroplaters. The information available is, however, insufficient for determining whether this is an exposure problem of national dimensions for the various source types, or whether the problem is dependent upon such local factors as discharge volume, degree of discharge control, and extent of receiving water volume. The extent of information on individual discharge monitoring and compliance status available for this assessment was insufficient to provide a basis for a quantitative exposure assessment, either in the nation as a whole or in local areas. In particular, the absence of these data prevents understanding the extent to which recent point source controls may have reduced or eliminated exposure risks.

Despite the above limitations, certain qualitative conclusions are possible concerning the risks to non-human biota. Where cyanide levels do exceed 5 to 7 μg/1, they will probably be in the vicinity of point discharge sources, and will diminish rapidly at increasing distances from the source. Hence, fish populations in surface waters are probably affected only over distances of several kilometers rather than over a broad geographic area. This type of exposure is exemplified by the fishkill incidents that have been associated with cyanide in the past. As far as terrestrial organisms are concerned, the risk from cyanide is not quantifiable, but appears to be considerably less than the risk to aquatic life.

7.2.1 Risk Considerations for Aquatic Organisms

Aquatic organisms may be exposed to scattered, highly localized risks as a result of cyanide in surface waters. Both acute and chronic effects on aquatic organisms may occur at cyanide concentrations in the range of 10 μg/1. Such concentrations have been observed infrequently at various locations throughout the United States. The fate analysis in this report indicates that the half-life of cyanides in surface water is relatively short; consequently, elevated concentrations in water would be expected only within a few kilometers of point-source dischargers. The potential aquatic risks may be associated with local batch-type discharges, even though the total annual discharges may not result in a sufficient loading to create observable steady-state concentrations. Dispersive emissions of cyanide, such as in road salt usage, are expected to contribute negligibly to high localized, ambient concentrations of cyanide compared with the cyanide contributions of point sources.

From an examination of reported fish kills attributed to cyanide, it appears that both isolated and recurring incidents of risk in fresh and estuarine waters can be associated with several types of man-made sources of cyanide. Reports of chronic recurrences in given locations appear to be somewhat more common in the pre-1976 period; however, reports compiled through 1977 indicate that episodes of cyanide-related fish kills still occurred, and in some locales, occasionally more than once a year. Twenty incidents were reported between 1972 and 1977.

In all, 16 reported fish kills were related to activities in the metals industry: 11 due to general metals production and 5 due to iron and steel production. Five other fish kills were reported to result from activities of the chemical industry, three from industrial discharges to POTWs, and one from mining activities. Three reported incidents were the result of unknown (or unreported) causes.

This distribution of incidents is not mutually exclusive, since several of the incidents were attributed to more than one of the activities cited. Though metal production, including iron and steel production, contributed to the majority of these incidents, it is evident that other activities have been associated with reported fish kills, and that in several instances, no suspected source of cyanide was found or reported.

Because of the paucity of data on cyanide exposure to saltwater organisms, it is difficult to assess the risks. However, the U.S. EPA's fish-kill records indicate a history of five reported kills of more than 30,000 fish, each attributed to cyanide in the estuarine headwaters of Chesapeake Bay near Sparrows Point, MD during 1972-73.

The materials balance presented in Section 3.1 indicates that industrial activities reportedly associated with cyanide-related fish kills are known to have created point-source (water) discharge of the pollutant. However, aquatic risks may be diminishing because the release of cyanide from some of these activities (e.g., iron and steel manufacture) is decreasing as a result of ongoing (NPDES) water pollution control initiatives. This may explain, for example, the absence of recent reports of fish kills from areas such as Sparrows Point, MD, where five large incidents were reported during 1972-73.

7.2.2 Risk Considerations for Terrestrial Organisms

The potential for exposure of terrestrial organisms to significant levels of cyanide appears to be small, although it is difficult to quantify. As indicated in Section 3.2.6, concentrations of cyanide in air are negligible in relation to the effect levels for mammals discussed in Section 4.1.5. Likewise, terrestrial mammals may be exposed to ambient surface water concentrations generally <0.1 mg/l. Consequently, it is extremely unlikely that these organisms will ingest lethal quantities of cyanide. For example, in order to ingest 10 mg/kg (which approximates the lethal oral dose given in Table 5-8, Section 4.1.5) an animal would have to drink 100 l/kg body weight. However, there have been incidences of cyanide poisoning in livestock that had consumed vegetative matter enriched with natural cyanides. The possibility for man-made contributions of cyanide to create similar situations remains unquantified.